LIPOPROTEIN CHANGES DURING NORMAL PREGNANCY AND PUERPERIUM

THESIS

FOR

DOCTOR OF MEDICINE

(MEDICINE)

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C E R T I F I C A T E

This is to certify that the work entitled "LIPOPROTEIN CHANGES DURING NORMAL PREGNANCY AND PUERPERIUM", which is being submitted as a thesis for M.D. (MEDICINE) Examination, 1998, Bundelkhand University, Jhansi, has been carried out by DR. ABDUL MAJEED MA in the department of Medicine, M.L.B. Medical College, Jhansi.

He has put in the necessary stay in the department as per university regulations.

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C O N T E N T

I N T R O D U C T I O N

INTRODUCTION

Pregnancy is a physiological state associated with tremendous changes in metabolism of carbohydrate, protein and fat. These changes are mediated by hormones mainly from placenta, anterior pituitary and adrenal cortex, though pancreas, thyroid and parathyroid hormones participate in adaptive changes that maintain the metabolic state in pregnancy. Of this, female sex hormones viz. oestrogen and progesterone have a key role in regulating the changes in lipid lipoprotein profile.

Sex hormones and lipid profile in female have received increasing attention because of the difference in the incidence of atherosclerosis between males and females i.e. in the reproductive age group females being comparatively protected than males.

Various studies have shown that in normal pregnancy there is an increase in serum total cholesterol and serum triglycerides, which progresses steadily till term and then abruptly falls after delivery (Boyd, 1934; Dieckman and Wegner, 1934; Watson, 1957).

At the same time, the deleterious effect of short term feeding of high cholesterol fat diet on the lipoprotein profile has been observed in several studies (Kannel et al, 1979). In the Indian context, it seems important because of the increased intake of total calories cholesterol and animal fat associated with pregnancy and

contd...2

lactation. This feeding behaviour persists for several months. The high prevalence of multiparity in India makes it more interesting.

On the other hand in high risk pregnancies like pre-eclampsia, eclampsia and intrauterine growth retardation, studies have shown a different pattern in lipoprotein profile. Boyd and Rochester (1935) found that concentration of plasma lipids varied greatly in eclamptic patients. According to De Alvarez (1961) there was an increase of serum total cholesterol in pre-eclampsia as compared with values in normal pregnancy. Studies from our department (Arora and Garg et al, 1989) also have shown a marked rise in serum total cholesterol and LDL cholesterol and a fall in HDL cholesterol in eclampsia as compared to normal pregnancy.

Thus noting the difference in lipoprotein response in normal and high risk pregnancy we were eager to know the exact role of lipoprotein in normal and high risk pregnancy and how changes in lipid profile can affect the maternal and foetal outcome.

The abrupt fall in lipoprotein levels that occurs immediately after delivery also raises some queries. How this raised lipoprotein disappears all of sudden ? Where does this lipoprotein go ?. Is it shifted to foetal circulation during labour ? Is it being utilised in uterine contraction during labour ? or is it shifted from maternal circulation to extravascular/subendothelial compartment ?

With this background a study "To study the lipoprotein changes during antenatal, natal and post natal period and cord blood of the new-borns in normal and high risk pregnancy and their effect on maternal and foetal outcome" was designed in our department. Towards this objective the present study was concentrated on normal pregnancy to evaluate lipoprotein changes and cord blood of their newborns. A parallel study concentrating on high risk pregnancy is in progress in our department.

The present study was aimed at the following objectives :

1. To know the trend of the changes in various lipoprotein fractions (STC, STG, HDL, LDL and VLDL) during antepartum, intrapartum and postpartum periods in normal pregnancy.
2. To know the lipoprotein pattern in the umbilical cord blood of newborns (from maternal and foetal end of the cord) and to compare these with that of maternal blood during intrapartum period.
3. To know the effect of parity over the lipoprotein changes in pregnancy and puerperium.
4. To know the effect of lactation on lipoprotein profile during puerperium.
5. To see the changes in lipoprotein pattern in relation to different mode of delivery i.e. vaginal delivery, elective or emergency caesarean section.

6. To see whether the outcome of pregnancy (live birth or still birth) have any relationship with maternal lipoprotein profile in an otherwise normal pregnancy.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Hyperlipidemia in normal pregnancy was reported by Bacqueral and Rodier as early as 1845. Virchow (1847) showed that the milky appearance of the sera of the pregnant women was due to the presence of fat as demonstrated by shaking the sera with ether, so that fat could be extracted. In 1911 Chaufford and associates demonstrated an increase of blood cholesterol in pregnant women by chemical methods.

Chiang and Yang et al (1955) in their study of lipid profile in normal pregnancy found that serum total cholesterol, triglycerides, LDL- , HDL-c were significantly elevated during second and third trimester of pregnancy but dropped sharply after delivery.

Udoh and Itam et al (1994) in their study on normal pregnant women showed a progressive increase in the serum total cholesterol and HDL-c throughout pregnancy and a decrease in their levels after delivery. In their study on 49 normal pregnant women STC rose from 154.60±14.99 mg/dl (3 months of pregnancy) to 214.99±19.25 mg/dl (9th month of pregnancy) followed by a decrease to 156.91±15.39 mg/dl at after one month of delivery. This represents a 39±11 per cent increase in STC at 9 months from the 3 months level. HDL-c showed an increase of 35±10 per cent at 9 months level.from 3 month level. The most significant month to month increase was recorded between the 6th and 7th month for both STC and HDL-c.

Darmandy and Postle (1982) in their study of lipid profile on 34 normal women monitored (STC & STG) before, during and after pregnancy. They found that all women displayed an initial fall in both substances during the first trimester. The concentration increased to maximum values during third trimester. There was a considerable variation in the time required for this hyperlipidemia to decline after delivery.

Sita Devi, Patrudu et al (1981) in longitudinal study of serum lipoprotein in normal pregnancy and puerperium estimated lipid profile in 23 pregnant women in three trimester and in the post natal period. Thirty three normal healthy non pregnant women of the same age group formed the control. A progressive rise was observed in serum triglyceride and cholesterol reaching the peak in the 3rd trimester and a definite fall in the postnatal period as compared to antenatal period. Among the hyperlipidemias the major one was type IV in the 1st trimester, type IIb and type IV in the second trimester and type IIb in the 3rd trimester and type IV again in the postnatal period. It was also noted that those who had an abnormal lipid profile in the 1st trimester dontinued to have abnormal lipid profile in the post natal period.

Mc Eachern and Gilmur determined whole blood cholesterol in twelve pregnant women and concluded that a marked elevation was found in about 30 percent of normal pregnant women, beginning about the 6th week prior to delivery and

that about 80 percent had a level above normal on the first day after delivery. The figures were still high on the 12th post partum day.

Oliver and Boyd (1955) after study of 12 normal primigravidae found that there was a highly significant rise in plasma ester and total cholesterol between 31st and 33rd weeks of pregnancy. By the 20th postpartum week these values had decreased considerably, but were all higher than the levels at the 12th week of pregnancy.

Dieckman and Wegner (1934) found that the total cholesterol to increase to 23 percent by term above the first trimester level and which decreased to 27 percent at the eighth post partum week from the values noted at term. This rise noted by Dieckman is considerably lower than De Alveraz et al (1959). Findings of 54 percent increase in 3rd trimester values above the first trimester values for total cholesterol and a 23 percent decrease in the values 6 to 7 weeks postpartum as compared to the 3rd trimester values.

Gardner and Gainsborough (1929) reported that free cholesterol increased during pregnancy to the 30th week with a decrease in ester cholesterol to about the same time. In their series, there occurs then a reversal of the curve so that at parturition, approximately a normal relationship exist again. Kaufman and Mihlbock (1933) did not notice these fluctuations but they reported little variation from the second month of gestation to term. Bugnard, Columbus and Guilheim Hinglais and Coverto (1940)

found an increase in total cholesterol in later months of pregnancy.

Tyler and Underhill (1925) determined whole blood cholesterol in pregnant and nonpregnant women of comparable age group, through out pregnancy starting from third month and found that cholesterol increase gradually till term, at that time it was roughly one third higher than that of three months.

Hermann and Neumann (1912) in their analysis of serum of pregnant women in various months of gestation concluded that during first 6-7 months, the serum total cholesterol might be increased and that during the last two months (32 cases) an increase in serum total cholesterol was the rule.

Plass and Tempkins (1923) also have given figures for the blood and lipids particularly cholesterol during pregnancy. These figures indicated a gradual rise from 4th month to term.

Arora and Kavita et al (1989) in their study showed that in normal as well as in toxaemia group STC, STG, HDL-c, VLDL and LDL-c showed a rising trend during antepartum period with a peak during labour followed by a fall in post partum period. Values were higher than the nonpregnant control group.

Arora and Vinita (1987) in their study showed STC level of 166.17±24.97 mg/dl in 1st trimester which reached to a peak of 263.44±39.8 mg/dl during labour. This decreased in the post partum period reaching 190.5±36.94 mg/dl at 1

month post partum.

Arora and Neeta et al (1993) studied the changes in lipoprotein profile in normal pregnancy and artificial termination of pregnancy (Elective/emergency LSCS). They observed that there was a rising trend in lipoprotein profile with a peak during labour followed by a fall in the post partum period, both in normal as well as artificial termination of pregnancy.

Mazurkiewicz, Watts et al (1994) in their study on serum lipid lipoproteins and apolipoproteins in pregnant non-diabetic patients reported that pregnant women had significant higher concentration of STG, STG, HDL-c, LDL-c, and apolipoprotein A and B, also that the ratio of STC:HDL cholesterol was not significantly different.

LIPID PROFILE IN UMBILICAL CORD BLOOD

Numerous investigators have shown that in maternal blood the concentration of cholesterol and phospholipids is greater than normal while in blood from umbilical cord at the time of birth is notably reduced.

Ortega, Gasper et al (1966) in their study on influence of maternal serum lipids and maternal diet during the 3rd trimester of pregnancy and umbilical cord blood lipids in two populations of Spanish newborns noted that a significant correlation was found to exist between maternal cholesterol concentration and those of newborn infants. A correlation was also found between maternal cholesterol levels and infant HDL-c and LDL-c levels. Further a

positive correlation was seen between maternal LDL-c and infant cholesterol and LDL-c. The relationship between maternal cholesterol and cord blood cholesterol was independent of participant's dietary, anthropometric and personal data. 3.1% of neonates showed total cord blood cholesterol concentration of 99.9 mg/dl. The mothers of these children showed the strongest concentration of cholesterol and LDL-c in the 3rd trimester of pregnancy, the shortest pregnancies and the smallest newborns of all subjects. Negative correlations were found between birth weight and cord blood cholesterol levels and LDL-c.

Heary and Kilby et al (1994) in their study on foetal and maternal lipoprotein metabolism in human pregnancy determined concentration and composition of lipid and apolipoprotein in maternal venous and umbilical arterial and venous blood. The objective of the study was toestablish whether the placenta has a role in foeto-maternal cholesterol metabolism through either synthesis or transplacental cholesterol flux. Study showed that pregnant women had raised levels of all lipid and lipoprotein fractions as compared with control subjects. In both umbilical venous and arterial blood concentration VLDL, LDL cholesteryl ester and triacylglycerols were lower than in maternal blood, but HDL-c levels were similar. There was no umbilical-arterio-venous differences in lipoprotein concentration or composition. This suggests that cholesterol synthesis or free cholesterol diffusions do not occur in the placenta.

Pontis et al (1979) studied antepartum and post partum lipoprotein levels in parturiting women and in umbilical cord blood of their newborns. The average values reported in umbilical cord blood were far low than that of maternal blood. The differences that exists between mother and baby in this respect varies from case to case and values prevailing in one seems to be entirely independent of those in other i.e. concentration of cholesterol is never same in mother and foetus. The differences have no constant or characteristic pattern.

Arora and Kavita et al (1989) in their study of the changes of lipid lipoprotein profile in normal pregnancy and toxaemia of pregnancy during antepartum and postpartum periods and in umbilical cord blood of their newborns, found that STC, HDL-c, STG, VLDL and LDL-c levels in umbilical cord blood were found to be very low in comparison to intra-partum values of the mothers. No difference in the levels of STC was found in newborns of normal pregnancy or toxaemia of pregnancy. HDL-c levels were higher in umbilical cord blood of newborns of normal pregnancy as compared to toxemia of pregnancy while converse was true with STG. However, differences in the levels of HDL and STG in normal pregnancy and toxemia of pregnancy were found to be statistically significant.

Boyd and Wilson (1934) studied exchange of lipid in the umbilical circulation at birth. They took samples of venous blood from the maternal end of the cord with the placenta still attached in utero. This represented the

venous blood entering the foetus. The contraction of uterus was assumed to have little effect upon the lipid content of venous blood, an assumption which was substantiated in part by the finding of similar results in cases of caesarean section in which the uterus was not contracted. It was later found that lipid concentration of venous blood slowly increases after is cord is clamped. They concluded that phospholipids, free cholesterol and cholesterol esters are added to umbilical blood between the time of delivery and the time of placental separation. Neutral fats may be either removed or added to umbilical blood by the placenta.

Konttinen et al (1964) studied serum lipids in normal pregnancy and pre-eclampsia and also umbilical cord blood of both groups. They concluded that cord samples of both the groups showed low levels of all the lipids studied and no differences were detectable between the two groups. The mean total cholesterol was about 80 mg/dl with a high content carried in alpha fractions. The serum triglycerides values were only about 1/8th of the values seen in their mothers, with no individual correlation between mother and child.

ROLE OF LACTATION OVER LIPID PROFILE DURING POST PARTUM PERIOD

Various investigators have shown that lactation affects the lipid profile in the postpartum period.

Erkkola and Viikari et al (1986) investigated serum lipid and lipoprotein fractions one day after delivery, 3 months later in lactating and non lactating mothers and 12

months later after initiation of menstruation in a group of 62 women. 29 of which formed a truly longitudinal group. STC decreased significantly within 3 months after delivery and a further significant decrease occurred during the following 9 months. LDL and HDL-c showed also a significant decrease within the postbpartal year. Serum triglycerides decreased within 3 months after delivery but no more significantly lower. In lactating mothers HDL-c:STC ratio was higher than in non lactating women. During luteal phase STC and LDL-c were lower and HDL-c:STC ratio was higher than earlier during the menstrual cycle. Data proved that pregnancy related changes in lipid metabolism did not wane within 3 months after delivery. They also showed that lactation affects lipid metabolism.

Darmandy and Postle et al (1982) in their study on lipid metabolism in pregnancy showed that there was a considerable variation in the time required for the hyperlipidemia of pregnancy to decline after delivery. Lactation appeared to be an important factor in this variation; women who bottle fed their infants maintained an elevated STG level for three times longer than those who breast-fed their infants.

Hermann and Neumann (1918) studied that whole blood cholesterol and total lipid decreased during normal lactation but remained elevated when lactation did not occur. According to Boyd (1935) the lipid concentration of blood plasma was found to decline consistently after delivery in

all cases where normal lactation occurred. The decline in values of plasma lipids during lactation was by and large due to loss of plasma neutral fat. After delivery the elevated STG concentration decreases rapidly and significantly greater utilization of STG in lactating women could be caused by the tissue specific direction of VLDL towards the mammary gland for milk synthesis (Darmandy et al, 1982). After neutral fat the greatest decrease was found in phospholipids and next in free cholesterol and ester cholesterol. Changes in cholesterol fraction were comparatively slight.

It appears that the phenomenon of lactation itself along with various metabolic and endocrine changes which accompany it, is chiefly responsible for the difference in the trends of plasma lipids values between lactating and nonlactating mothers. Whether the blood plasma lipids falls during lactation because they are secreted in milk or because of the presence or absence of certain hormones or other effects are not yet clear. But some investigators have tried to correlate these effects with the activity of lipoprotein lipase enzyme. The enzyme activity is raised in adipose tissue during early pregnancy in experimental animals and subsequentlt falls as gestation progresses. These changes are specific for the adipose tissue enzyme. After delivery, the elevated STG concentration decreases rapidly and the significantly greater utilisation of STG in lactating women could be caused by the tissue specific

direction of VLDL towards the mammary gland for milk synthesis. Evidence to support this hypothesis is provided by studies on postpartum rats (Hamosh et al, 1970). Lipoprotein lipase activity is depressed in adipose tissue but greatly elevated in mammary gland from suckling but not from non-suckling rats. The STG concentration in suckling rats returns to normal within a few hours of delivery, but remains high for over 18 hours in non-suckling rats. It is possible that the comparable pattern between lactating and non-lactating mothers in this study could have a similar explanation (Darmandy et al, 1982).

Several explanations have been offered as to where the excessive amounts of blood lipids go once pregnancy is over, it is proposed that in puerperium blood lipids are discharged through bile (Bac eisler and Herver, 1914). Urine and faeces (Hermann and Neumann, 1912) and in milk. According to Boyd and most of these theories, fat content of blood is static value. These theories appear to take for granted that if a fixed amount of blood lipids are removed from blood by the liver, kidney, intestinal tract etc, the puerperium, this removal would cause a lowering of the concentration of blood lipids. Blood lipids are continuously in equilibrium with tissue lipids, if lipids are removed from blood, the loss is made up by the addition of lipids from the fat depots. It is unlikely that any removal except a very excessive one such as occurs in the increased metabolic rate of high fever (Boyd, 1935) could be itself account for lowering of the values of blood lipids. By

knowing the factors which influence the equilibrium between blood and tissue lipids. It will be possible to explain the lipid changes in pregnancy and lactation. Boyd (1935) observed that in the puerperium this equilibrium is altered in the direction of lowering of the level of plasma lipids, but if normal lactation is prevented, the change is inhibited or reversed.

Pontis, Gupte and Purandare (1978) noted a significant fall in cholesterol level from antepartum to postpartum period. This denotes that the placenta is the principal organ responsible for the elevation of cholesterol during pregnancy. It is also true that the hormonal levels which are affected during pregnancy are changed with eviction of placenta. Therefore, these levels may indirectly be for the decline in lipid parameters. It has been stated by Berzin and Vonstudintz (1957) that oestrogen caused rise in circulating lipid levels. Both oestrogen and progesterone are of placental origin. So the placenta itself is the primary cause for the elevation of lipids which slowly returns to pregestational levels after expulsion of placenta.

EFFECT OF DIET IN SERUM LIPOPROTEIN PROFILE

In 1929, Gardner and Gainsborough carried out complete study on cholesterol metabolism and concluded that during period of fasting, cholesterol content of plasma varies markedly in different healthy persons but is fairly constant in subjects. A single meal does not cause any change but prolonged diets high or low in sterol will cause

variation in cholesterol. The free cholesterol remains fairly constant but cholesterol esters shows greatest change..

According to Mullick and Bagga (1964), in healthy females, serum lipids and its fractions vary with the nutritional status which is itself dependent upon the socio-economic conditions of individual. Values for high income groups are close to those reported by Boyd. In pregnant females the increase in serum total lipids occurring in the first eight weeks of 1st trimester was more marked in the vegetarians than in the non-vegetarians. STC, ester cholesterol and free cholesterol showed the reverse trend. In the 2nd trimester this difference was narrowed. In the 3rd trimester there was no difference in the serum total lipids between vegetarians and non-vegetarians, but there was now a slight increase for the non-vegetarians. Thus, diet has no significant influence on lipid synthesis in the later period of pregnancy.

Green (1966) determined total cholesterol serially in a group of young women before and during pregnancy while they consumed their usual diet or a fat modified diet known to have a hypocholesterolemic effect. During 1st trimester of pregnancy there was a slight but definite fall in serum cholesterol levels. After the 1st trimester serum cholesterol levels increases gradually to peak at or near term. These changes occur both in normal and hypercholesterolemic females and is not affected by fat modified diets.

Hansen and co-workers studied 80 pregnant women and found no significant correlation between mothers' intake of colories, proteins, fat and fatty acids to serum cholesterol or fatty acid levels during 3rd trimester.

Moses and his colleagues studied 65 young pregnant women from 5th month of pregnancy to term. 35 received ordinary institutional diet while 30 received same diet with 2 gm daily supplement of cholesterol. There was no significant difference in serum lipids of these two groups.

Arora and Vinita (1987) studied the influence of dietary fat on serum total cholesterol level during antepartum, intrapartum and postpartum periods of pregnancy and in the cord blood of newborns. They concluded that the levels of STC were higher in subjects taking high fat diet and lower in those taking normal and low fat diets with advancement of pregnancy during labour, after delivery and in late postpartum period values were not statistically significant. However, the cord blood STC values in relation to fat diet of mother in third trimester were highly significant.

HORMONAL CHANGES DURING PREGNANCY AND POSTPARTUM PERIOD

Almost every endocrine tissue participates in adaptive changes that maintain the metabolic state of the mother during normal pregnancy. In this respect placenta anterior pituitary and adrenal cortex holds a key role, although thyroid, parathyroid and pancreas show distinct physiological changes during pregnancy leading to increase in output of respective hormones. At 6-8 weeks of pregnancy

there is transfer of functions of corpus luteum to the placenta which acts temporarily as a new endocrine organ.

1. PLACENTAL HORMONES

During pregnancy, ovarian steroid production wanes, and continued production of oestrogen and progesterone then depends primarily on their synthesis in the placenta.

a. Oestrogens

Oestriol is the main pregnancy oestrogen which accounts for 80-90% of oestrogen formed in late pregnancy. It has modest biological activity relative to oestradiol. The predominant oestrogen secreted by non-gravid females. Urinary oestrogen rises progressively during pregnancy and at term it is about 40-50 mg/dl per 24 hour urine. Oestrogen levels fall significantly within 3 days and reach a basal level by 7th post partum day.

Eilert (1949) found that oestrogen administered to women evoked an increase in the plasma total lipids.

Russ and associates (1955) found that the administration of oestrogen lowered the beta lipoprotein but raised the alpha-lipoproteins.

b. Progesterone

After the first trimester, the placenta becomes capable of producing sufficient progesterone to maintain gestation. At term placenta may secrete approximately 250 mg progesterone daily. Progesterone levels in maternal

blood increases progressively with gestation. Cholesterol derived from the maternal blood is the main substance for the trophoblastic synthesis of progesterone (Ronald and Kalkhoff et al, 1978). Progesterone levels fall significantly within 3 days, reach at basal level by 7th postpartum day. In non-lactating females it again rises by 21st day.

Oestrogen and progesterone levels remain low in lactating females.

c. Human Chorionic Gonadotropin (HCG)

Concentration of HCG rises to peak values by 8-12 weeks of gestation. Thereafter there is a decrease in HCG levels to a plateau that is maintained throughout the remaining of pregnancy. It becomes undetectable in urine by 7-10 days postpartum. Smith and Smith (1935) claimed that HCG are increased in serum&urine in pre-eclamptic toxaemia.

d. Human Chorionic Somatomammotropin (HCS or HPL)

A polypeptide hormone secreted by placenta gradually increases and eventually reaches a maximum of 5-8 microgram/ml at term. At the end of the pregnancy its production rate has been estimated to be about 1 gm/day. After delivery of placenta, maternal blood concentration return to undetectable levels within 24 hours. Although secretion of HPL has been considered to be autonomous recent findings suggest that maternal plasma glucose and/or free fatty acid may modulates its secretion.

2. ANTERIOR PITUITARY HORMONES

a. Human Growth Hormone(HGH)

Basal concentration of HGH are low during early pregnancy and do not change remarkably with advancing gestation.

b. Prolactin

Prolactin in pregnancy begins to increase approximately 30 days after the mid menstrual cycle peak of leutinising hormones. Rising prolactin levels continue to increase to reach peak levels at term. Serum prolactin declines rapidly after parturition, if the women does not breast fed. However, prolactin levels increase sharply with breast feeding episodes. After several months of lactation prolactin concentration decreases to non pregnant values.

3. ADRENAL GLUCOCORTICOIDS

Cortisol metabolism is significantly altered during pregnancy and maternal plasma cortisol level rises progressively throughout gestation. The plasma levels of transcortin also rises progressively to a peak in 3rd trimester. The elevated transcortin level is mainly or entirely secondary to the increased plasma oestrogen concentration. The maternal tissues are exposed to an average daily concentration of cortisol that is more than twice normal.

4. THYROID HORMONES

Thyroxin : Like oestrogens it depressed the blood lipid levels. Patterson, Hund and Nicodeus (1938) believed that hypercholesterolemia of pregnancy is due to subclinical hypothyroidism.

Lister (1955) and Russel (1956) found that protein bound iodine and serum precipitate iodine are elevated as early as 2nd month of pregnancy. These levels have been found to reach as high as those seen in individuals with overt hyperthyroidism.

Slrisower (1958) found that the thyroid hormone depresses serum lipids but during pregnancy the tissues become more refractory to the effect of thyroxin.

5. PANCREATIC HORMONES

Insulin : The basal levels of insulin tend to become progressively higher as term gestation is approached. Also a much greater amount of insulin is released in response to glucose stimulation. However, during pregnancy a state of insulin resistance exists. Insulin has a pronounced antilipolytic effect and antagonises the lipolytic effect of hormones mainly by inhibiting the hormone sensitive lipase in the adipose tissue. Thus, it reduces the release of not only free fatty acids but also glycerol.

MATERIAL AND METHODS

MATERIAL AND METHODS

Present study was carried out in the Departments of Medicine and Obstetrics & Gynaecology, M.L.B. Medical College, Hospital, Jhansi.

Women with normal pregnancy attending the antenatal clinic and admitted in wards of Obstetrics and Gynaecology were included in the study. Subjects suffering from diseases which are likely to have an altered lipoprotein levels such as coronary artery disease, renal disease, liver disease, diabetes mellitus were excluded from the study.

A total of 104 cases of normal pregnancy were enrolled in this study. Of which only 60 cases were taken for final assessment. The remaining being dropped at various stages of study due to lack of sufficient follow up. Cases who developed complications of pregnancy like pre-eclampsia, eclampsia or gestational diabetes during follow up were also excluded.

After getting the particulars of the subjects like age, address and occupation, details regarding following points were noted.

OBSTETRIC HISTORY

Obstetric history included following points :

- Date of last menstrual period.
- Gravidae and parity.
- Pregnancy induced hypertension, pre-eclampsia, eclampsia, IUGR, and prematurity during previous pregnancy.

MEDICAL HISTORY

Medical history included hypertension, renal disease, liver disease, diabetes mellitus, hypo/hyperthyroidism.

FAMILY HISTORY

This included hypertension, diabetes mellitus and coronary artery disease.

PERSONAL HISTORY

This included smoking, alcohol, tobacco chewing etc. and nature of contraceptive used, if any.

TREATMENT HISTORY

Therapy with corticosteroid, thiazides and betablockers.

LACTATIONAL STATUS

DIETARY HISTORY

Average daily intake of calories and fat was assessed during each visit. Type of diet (vegetarian/ non-vegetarian) was also noted.

General, systemic and obstetric examinations with special reference to blood pressure, oedema and weight gain were done in each visit.

INVESTIGATIONS

Urine albumin, sugar, microscopy and blood haemoglobin, blood sugar (fasting and postprandial) were done in each visit.

Investigations	Trimester I	Trimester II	Trimester III	Intra-partum	Postperum period 24h	Postperum period 7th d	Postperum period 1 month
Urine: Albumin							
Sugar							
M/E							
Haemoglobin							
Blood sugar :							
Fasting							
Postprandial							

Lipoprotein estimation was done at the Lipid Research Laboratory attached to Department of Medicine. Estimations were done during 1st, 2nd and 3rd trimester, of pregnancy, during labour (Intrapartum - IP), within 24 hour postpartum (24h PP), 7th day postpartum (PP7d) and one month postpartum (PP1m). Cord blood lipoprotein profile (placental and foetal side) were also noted immediately after delivery.

Serum total cholesterol (STC), serum triglycerides (STG) and high density lipoproteins (HDL) were estimated by standard methods. Very low density lipoprotein(VLDL) and low density lipoprotein (LDL) were calculated by the following formulae :

VLDL (mg/dl) = STG/5 (This is valid upto the value of $\lesssim$300 mg/dl).

LDL (mg/dl) = STC - (STG/5 + HDL)

= STC - (VLDL + HDL)

Lipoprotein Profile

Period	STC	STG	HDL	LDL	VLDL	LDL/HDL
1st trimester						
2nd trimester						
3rd trimester						
Intrapartum						
24hrs postpatum						
7 days "" ""						
1 month "" ""						
Umbilical cord blood :						
Placental side						
Foetal side						

To note the effect of lactation on lipoprotein profile during puerperium, the cases were divided into two groups (Lactating and non-lactating). Lipoprotein profile between these two groups were compared at past-partum, 7th day and postpartum 1 month.

To know the lipoprotein changes in relation to mode of delivery the subjects were divided into 3 groups :

Group I : Subjects who had vaginal delivery.

Group II : Subjects undergone emergency caesarean section.

Group III : Subjects undergone an elective caesarean section.

Lipoprotein profile between these three groups were compared during intrapartum, 24 hrs postpartum, 7 days postpartum and 1 month postpartum.

It was also tried to compare the lipoprotein changes in relation to pregnancy outcome (live birth Vs still birth) in subjects having an otherwise normal pregnancy till term. 52 subjects who delivered live baby were denoted as group A and 7 patients who had still birth (Fresh still birth) were taken as group B.

To note the effect of parity on lipoprotein profile, the subjects were also divided according to parity i.e. primigravidae (22 cases)and multigravidae (38 cases).

The lipoprotein pattern in these two groups was compared at 1st, 2nd and 3rd trimester, intrapartum, and post partum 24 hours, 7 days and 1 month.

O B S E R V A T I O N S

OBSERVATIONS

In the present study 60 cases were taken for the final analysis. General characteristics of the cases were as follows :

Age :	Range	: 17 to 36 years
	⩽20 years	: 4 (6.67%) cases
	20-30 years	: 53 (88.33%) cases
	⩾30 years	: 3 (5.00%) cases
Parity :	Primi para	: 22 (37.67%) cases
	Multi para	: 38 (63.33%) cases
Socio-economic status :		
	Low	: 32 (53.33%) cases
	Middle	: 28 (46.67%) cases
Dietary habit:	Vegetarian	: 36 (60.00%) cases
	Non-vegetarian	: 24 (40.00%) cases

Lactating Status :

In the present study out of total 60 cases, only 38 cases were analysed for lactatory status.

Lactating	: 29 (76.31%) cases
Non-lactating	: 9 (23.69%) cases

None of the subject was habituated to smoking, alcohol or tobacco. None was taking oral contraceptives.

GRAPH 01

Serum Total Cholesterol levels (mean) at different stages of pregnancy puerperium

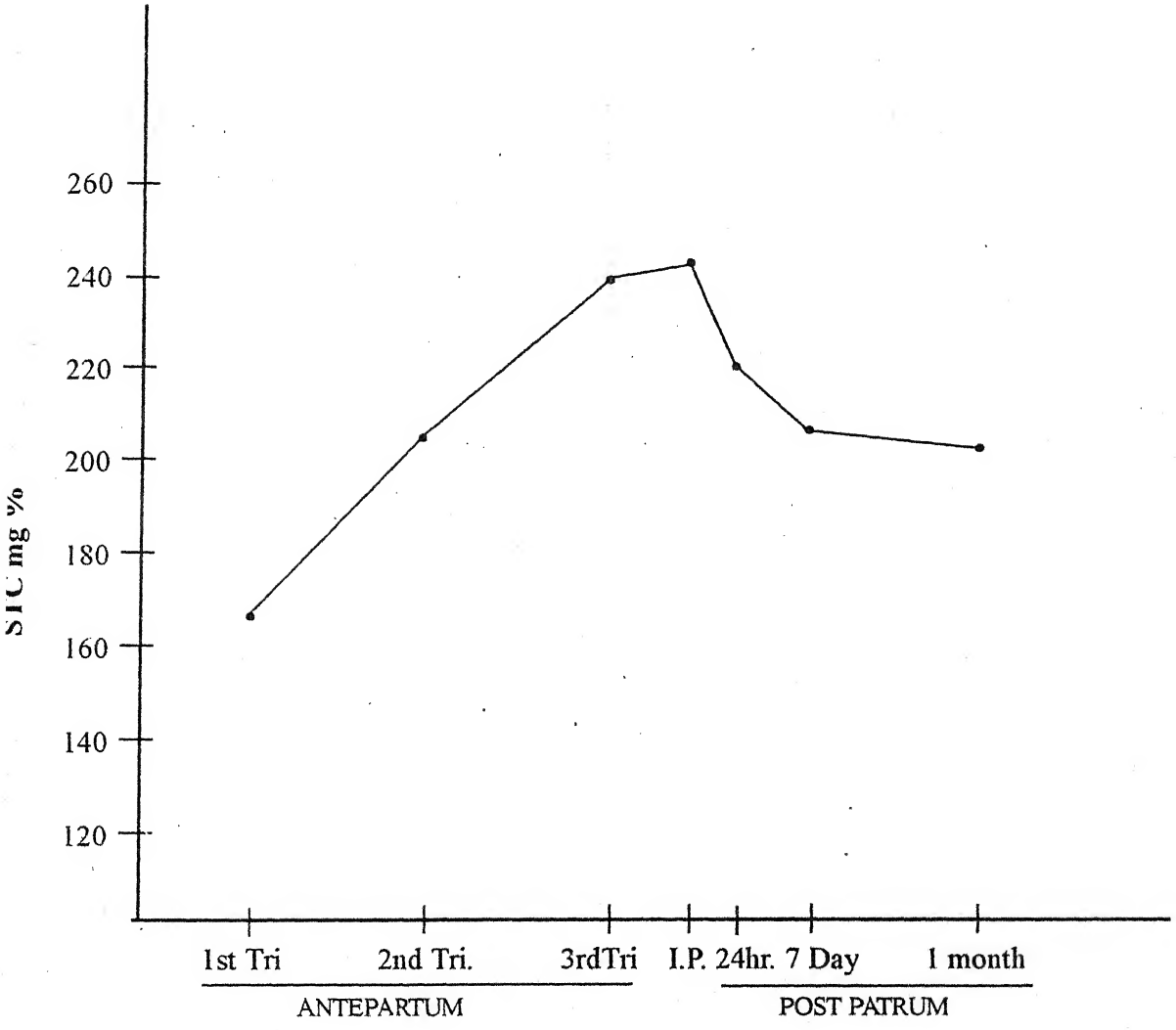


TABLE I : Showing mean values of STC during antepartum, intrapartum and postpartum periods. (Mean±S.D., mg/dl).

	Trimester			Intra-partum	Post partum		
	I	II	III		24hr	7 day	1month
n	32	38	46	56	47	32	28
STC	168.78 ±33.01	217.40 ±25.10	240.15 ±22.11	243.07 ±25.72	222.10 ±26.52	211.40 ±20.44	206.14 ±26.12

Table I shows that STC levels raised from 168.78± 33.01 mg/dl at 1st trimester to 217.20±25.10 mg/dl at 2nd trimester and further increased to 240.15±22.11 mg/dl at 3rd trimester reaching a peak value of 243.07±25.72 mg/dl during labour. The STC level declined to 222.10±26.52 mg/dl at 24 hours post partum. The decrease was further occurred to 211.40±20.44 mg/dl and 206.14±26.12 mg/dl at 7 days and 1 month of post partum respectively. But STC level was still higher (206.14±26.12 mg/dl) than the level of 1st trimester (168.78±33.01 mg/dl).

Statistical Analysis

p $>$ 0.05 Not significant

p $<$ 0.05 Significant

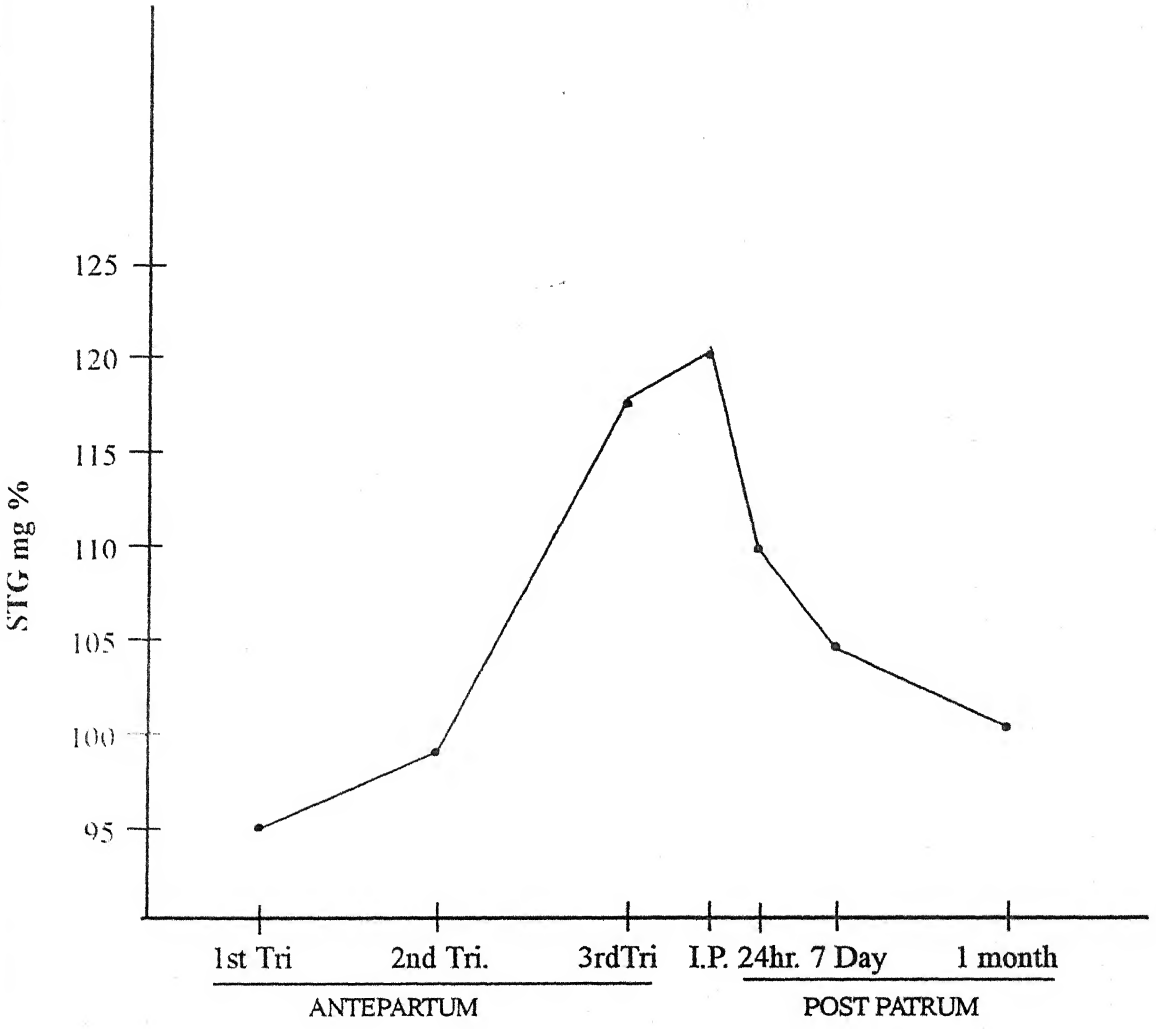
p $<$0.01 & $<$0.001 Highly significant

N = Number of subjects considered for statistical analysis.

While comparing between two groups only those subjects who had representation in both groups, were considered for statistical analysis. Number of cases was denoted as 'N'.

GRAPH 02

Serum Triglyride (STG) levels (mean) at different stages of pregnancy and puerperium



Group compared	'N'	't' value	'p' value
I Vs II	26	7.23	<0.001
I Vs III	28	13.1	<0.001
I Vs IP	29	14.3	<0.001
I Vs PP24h	24	8.3	<0.001
I Vs PP7d	24	7.91	<0.001
I Vs PP1m	24	7.15	<0.001
II Vs III	33	5.9	<0.001
II Vs IP	37	6.99	<0.001
II Vs PP24h	31	1.72	>0.05
IP Vs PP24h	44	9.6	<0.001
IP Vs PP7d	31	11.6	<0.001
IP Vs PP1m	26	12.2	<0.001

TABLE II : Showing mean values of STG during antepartum, intrapartum and post-partum periods (Mean$\pm$S.D., mg/dl).

	Trimester			Intra-partum	Post partum		
	I	II	III		24hr	7 day	1month
n	32	38	46	56	47	32	28
STG	95.41 $\pm$10.25	98.72 $\pm$13.73	118.77 $\pm$18.11	120.78 $\pm$17.23	110.48 $\pm$13.09	103.18 $\pm$13.42	102.96 $\pm$15.70

Serum Triglycerides (STG) raised from 95.41$\pm$10.25 mg/dl at 1st trimester to 98.72$\pm$13.73 mg/dl at 2nd trimester. STG level further increased to 118.77$\pm$18.11 mg/dl at 3rd trimester reaching a peak value of 120.78$\pm$17.23 mg/dl during labour. Then STG level declined to 110.48$\pm$13.09 mg/dl

within 24 hours of delivery. Further decreased occurred to 103.18±13.42 mg/dl and 102.96±15.7 mg/dl at 7 days and 1 month post partum respectively. The level of STG at 1 month was still higher than that of at 1st trimester.

Statistical Analysis

Group Compared	'N'	't' value	'p' value
I Vs II	26	2.25	<0.05
II Vs III	28	6.48	<0.001
I Vs IP	29	6.90	<0.001
I Vs PP24h	24	4.2	<0.001
I Vs PP7d	24	3.31	<0.01
I Vs PP1m	24	2.83	<0.05
II Vs III	33	4.33	<0.001
II Vs IP	37	4.91	<0.001
IP Vs PP24h	44	3.31	<0.01
IP Vs PP7d	31	5.04	<0.001
IP Vs PP1m	26	5.21	<0.001

Statistical analysis showed that the rise in STG level from 1st trimester to 2nd trimester was statistically significant while the value at 3rd trimester and intrapartum period when compared to that of 1st trimester values were highly significant. The STG level dropped from intrapartum within 24 hours of delivery and this decrease was statistically significant. The difference between STG level of 1 month post partum and 1st trimester was also significant.

GRAPH 03

HDL - Cholesterol levels (mean) at different stages of Pregnancy and Puerperium

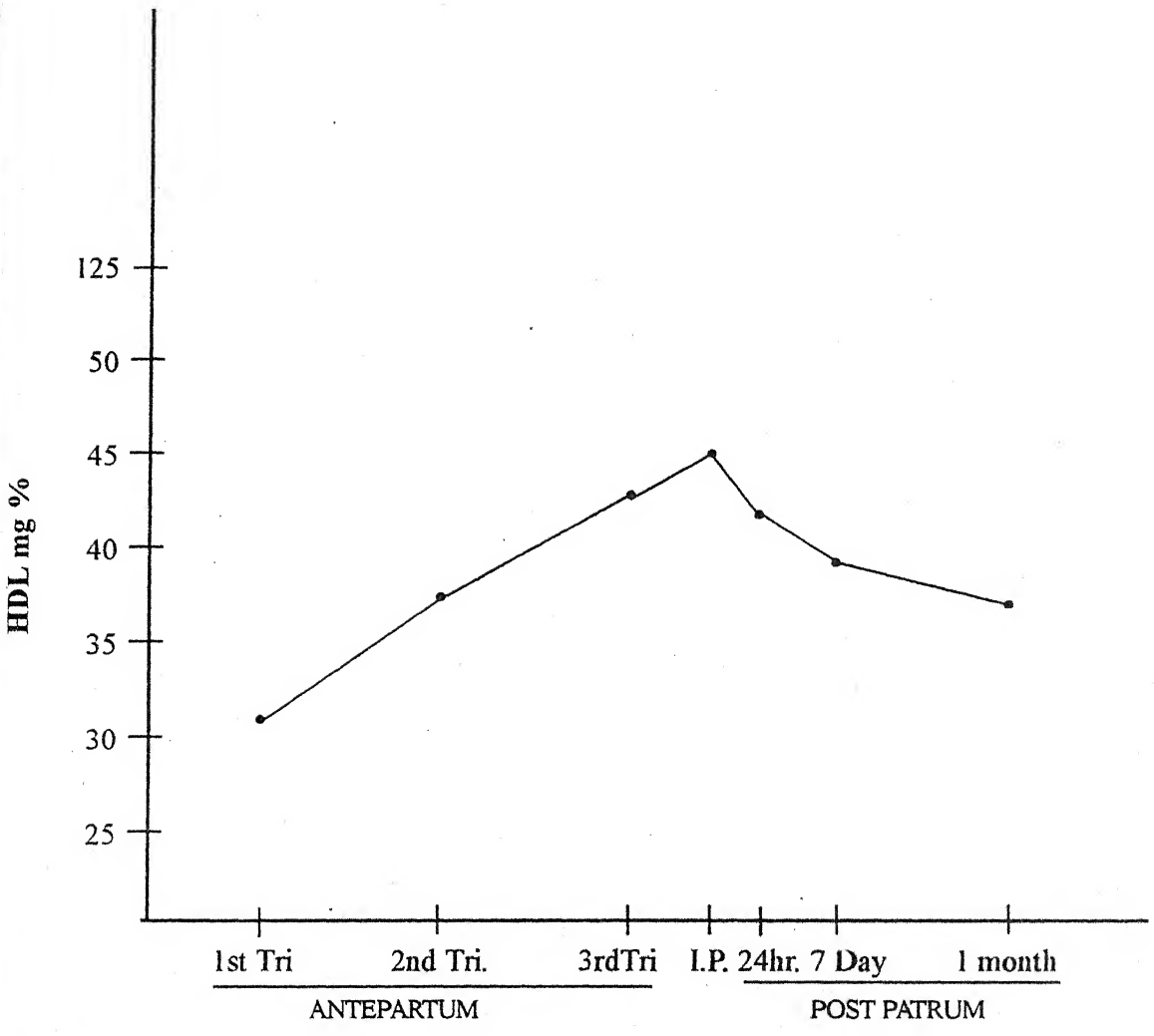


TABLE III : Showing HDL levels during antepartum, intrapartum and postpartum periods (Mean±S.D., mg/dl).

	Trimester			Intra-partum	Post partum		
	I	II	III		24hr	7 day	1month
n	32	38	46	56	47	32	28
HDL	31.01 ±3.61	37.72 ±5.19	43.95 ±6.70	44.58 ±7.33	42.29 ±5.98	39.59 ±4.25	37.28 ±4.48

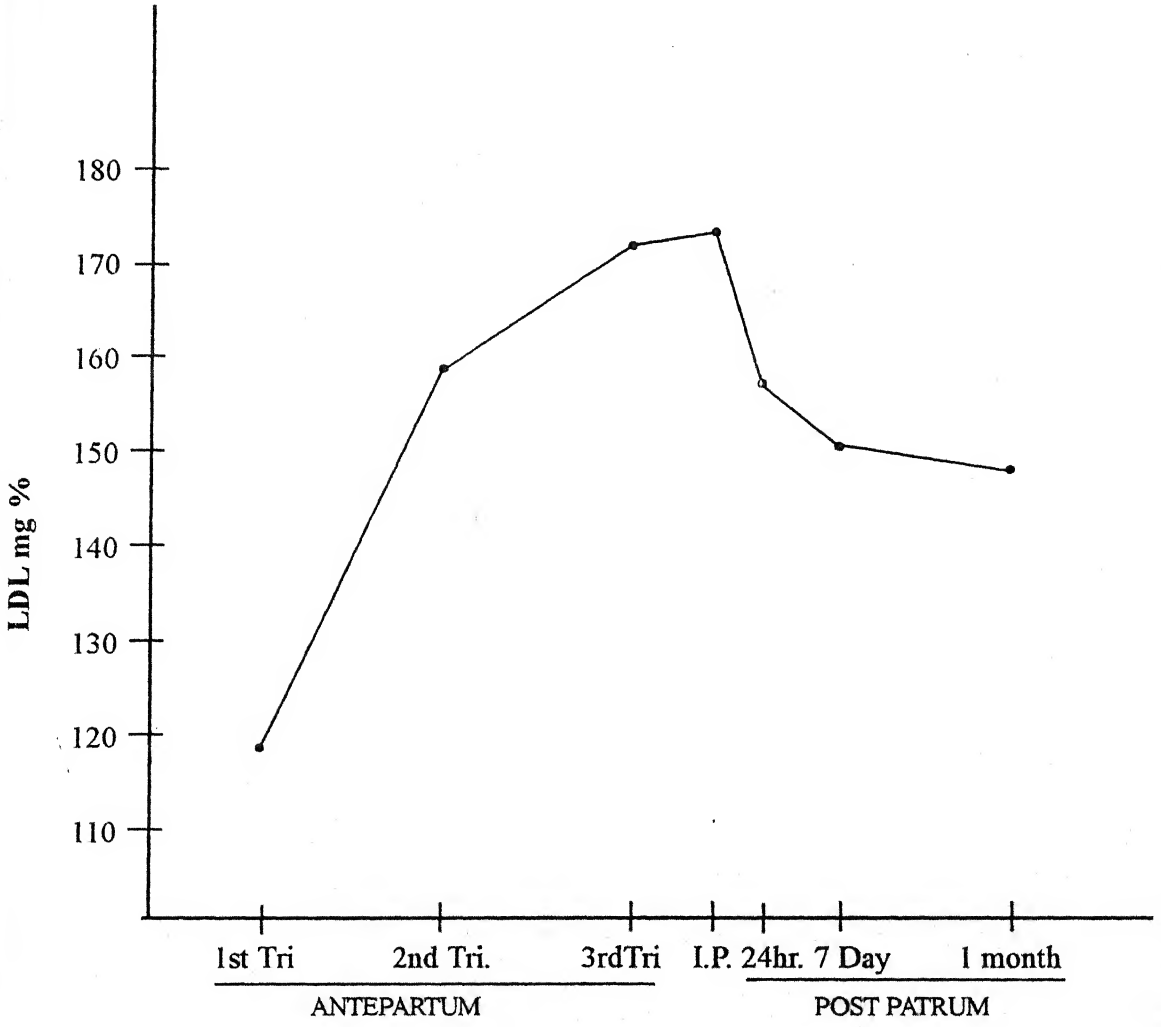
Table III shows that HDL values raised from 31.01±3.61 mg/dl at 1st trimester to 37.72±5.19 mg/dl at 2nd trimester. It further increased to 43.95±6.70 mg/dl at 3rd trimester. It reached at a peak value of 44.58±7.33 mg/dl during labour. The HDL levels then declined to 42.29±5.98 mg/dl within 24 hours postpartum which further decreased to 39.59±4.25 mg/dl and 37.28±4.48 mg/dl at 7 days and 1 month postpartum respectively. The values of HDL at 1 month post partum (37.28±4.48 mg/dl) was still higher than 31.01±3.61 mg/dl at 1st trimester.

Statistical Analysis

Group compared	'N'	't' value	'p' value
I Vs II	26	10.35	<0.001
I Vs III	28	14.1	<0.001
I Vs IP	29	15.8	<0.001
I Vs PP24h	24	13.2	<0.001
I Vs PP7d	24	10.2	<0.001
I Vs PP1m	24	6.9	<0.001
II Vs III	33	6.1	<0.001
II Vs PP24h	31	5.1	<0.001

GRAPH 04

LDL - Cholesterol levels (mean) at different stages of Pregnancy and Puerperium



II Vs PP1m	24	0.06	>0.05
IP Vs PP24h	44	2.02	<0.05
IP Vs PP7d	31	2.91	<0.01
IP Vs PP1m	26	3.93	<0.001

Statistical analysis shows that rise in HDL level in 1st trimester to 2nd trimester was statistically highly significant. The rise from 2nd trimester to 3rd trimester and also from 2nd trimester to intrapartum was highly significant. Significant drop in HDL levels was noticed after delivery within 24 hours postpartum period. Further fall occurred by 7 days postpartum and 1 month postpartum. The fall being highly significant when compared to the intrapartum levels.

TABLE IV : Showing values of serum LDL cholesterol during antepartum, intrapartum and post partum periods (Mean$\pm$S.D., mg/dl).

	Trimester			Intra-	Post partum		
	I	II	III	partum	24hr	7 day	1month
n	32	38	46	56	47	32	28
LDL	118.84 $\pm$30.86	159.10 $\pm$20.12	172.33 $\pm$19.43	172.55 $\pm$20.79	157.43 $\pm$24.70	151.17 $\pm$18.50	148.35 $\pm$22.30

Table IV shows that LDL values raised from 118.84$\pm$ 30.86 mg/dl at 1st trimester to 159.10$\pm$20.12 mg/dl at 2nd trimester followed by a further rise to 172.33$\pm$19.43 mg/dl at 3rd trimester. Values reached to at peak level of 172.55$\pm$20.79 mg/dl at intrapartum. The LDL levels then declined to 157.43$\pm$24.70 mg/dl within 24 hours postpartum.

Further decrease occurred to 151.17±18.50 mg/dl and 148.35 ±22.30 mg/dl at 7 days and 1 month post partum period respectively. The LDL level at 1 month postpartum (148.35 ±22.30 mg/dl) was still higher than the 1st trimester value(118.84±30.86 mg/dl).

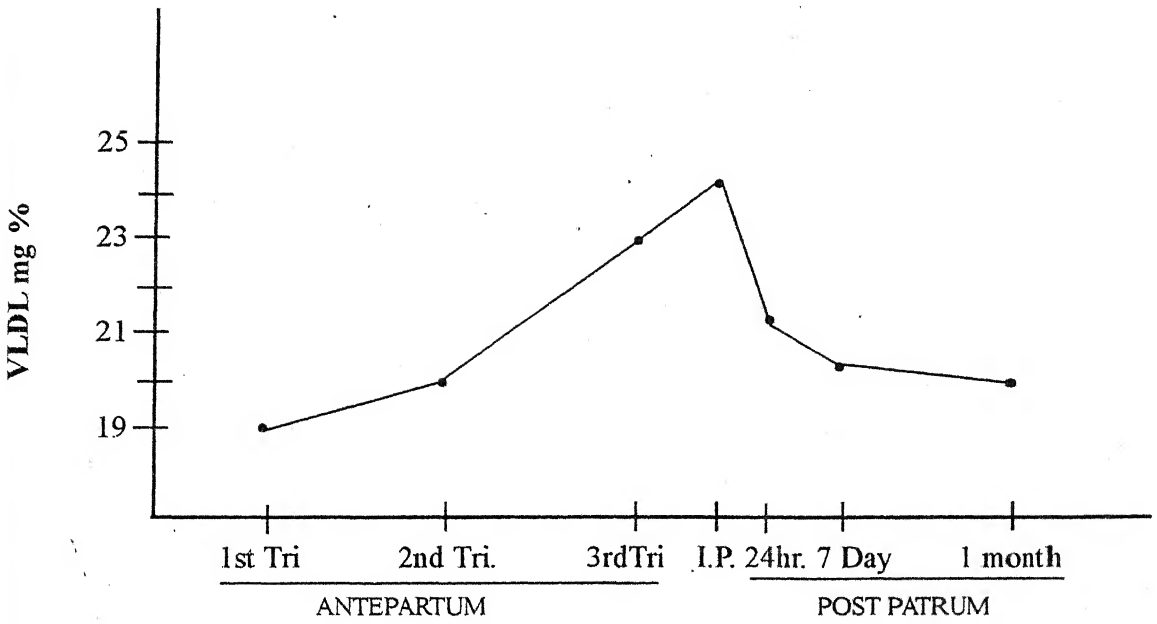
Statistical Analysis

Groups compared	'N'	't' value	'p' value
I Vs II	26	5.75	<0.001
I Vs III	28	12.4	<0.001
I Vs IP	29	12.5	<0.001
I Vs PP24h	24	5.01	<0.001
I Vs PP7d	24	5.23	<0.001
I Vs PP1m	24	5.38	<0.001
II Vs III	33	2.16	<0.05
II Vs IP	37	2.32	<0.05
III Vs IP	52	0.12	>0.05
IP Vs PP24h	44	5.80	<0.001
IP Vs PP7d	31	6.10	<0.001
IP Vs PP1m	26	7.20	<0.001

Statistical analysis shows that rise in LDL level from 1st trimester to 2nd trimester was statistically highly significant while the rise from 2nd trimester to 3rd trimester and 2nd trimester to intrapartum were statistically significant. The rise in LDL from 3rd trimester to intrapartum was statistically insignificant. Significant

GRAPH 05

VLDL - [illegible] levels (mean) at different stages of Pregnancy and Puerperium



drop in LDL cholesterol was noticed after delivery within 24 hour postpartum. The values at 7 days and 1month post partum were statistically highly significant when compared with that of intrapartum. The LDL level at 1 month post partum was higher than that of 1st trimester and the difference was statistically significant.

TABLE V : Showing the mean values of VLDL during antepartum, intrapartum and postpartum periods (Mean±S.D., mg/dl).

	Trimester			Intra-	Post partum		
	I	II	III	partum	24hr	7 day	1month
n	32	38	46	56	47	32	28
VLDL	19.08 ±2.02	20.20 ±2.66	23.75 ±3.38	24.15 ±3.42	21.65 ±2.87	20.63 ±2.62	20.53 ±2.99

Table V shows that VLDL values raised from 19.03± 2.02 mg/dl at 1st trimester to 20.20±2.66 mg/dl at 2nd trimester followed by a further rise to 23.75±3.38 mg/dl at 3rd trimester reaching a peak value of 24.15±3.42 mg/dl during labour. The VLDL then declined to 21.65±2.87 mg/dl within 24 hours postpartum. A further decrease occurred to 20.63±2.62 mg/dl and 20.53±2.99 mg/dl at 7 days and 1 month post partum respectively. The VLDL level at 1 month postpartum (20.53±2.99 mg/dl) was still higher than the calues at 1st trimester (19.08±2.02 mg/dl).

Statistical Analysis

Groups compared	'N'	't' value	'p' value
I Vs II	26	2.27	<0.05
I Vs III	28	7.05	<0.001

I Vs IP	29	9.81	<0.001
I Vs PP24h	24	3.61	<0.01
I Vs PP1m	24	1.9	>0.05
II Vs III	33	6.06	<0.001
II Vs IP	37	9.08	<0.001
III Vs IP	52	2.12	<0.05
IP Vs PP24h	44	3.71	<0.01
IP Vs PP7d	31	4.68	<0.001
IP Vs PP1m	26	4.69	<0.001

Statistical analysis shows that the VLDL level at 2nd trimester was statistically significant when compared to that of 1st trimester. The values at 3rd trimester and intrapartum period became highly significant when compared with that of 1st trimester. Statistical significant values were also noted between 2nd Vs 3rd trimester and 3rd trimester Vs intrapartum. A significant drop in VLDL occurred after delivery within 24 hours of postpartum when compared with that of intrapartum period. The VLDL level at 1 month postpartum was higher than that of 1st trimester but the difference was statistically insignificant.

Table VI depicts that the LDL : HDL ratio shows a slight increase from 3.83 ± 0.56 at 1st trimester to 4.18 ± 0.98 in 2nd trimester. Then it decreased to 3.96 ± 0.51 at 3rd trimester. The ratio then remained without much change i.e. 3.98 ± 0.82 during labour, 3.87 ± 0.82, 3.83 ± 0.63 and 3.95 ± 0.61 at 24 hour, 7 days and 1 month postpartum respectively.

GRAPH 06

LDL : HDL - Ratio at different stages of Pregnancy and Puerperium

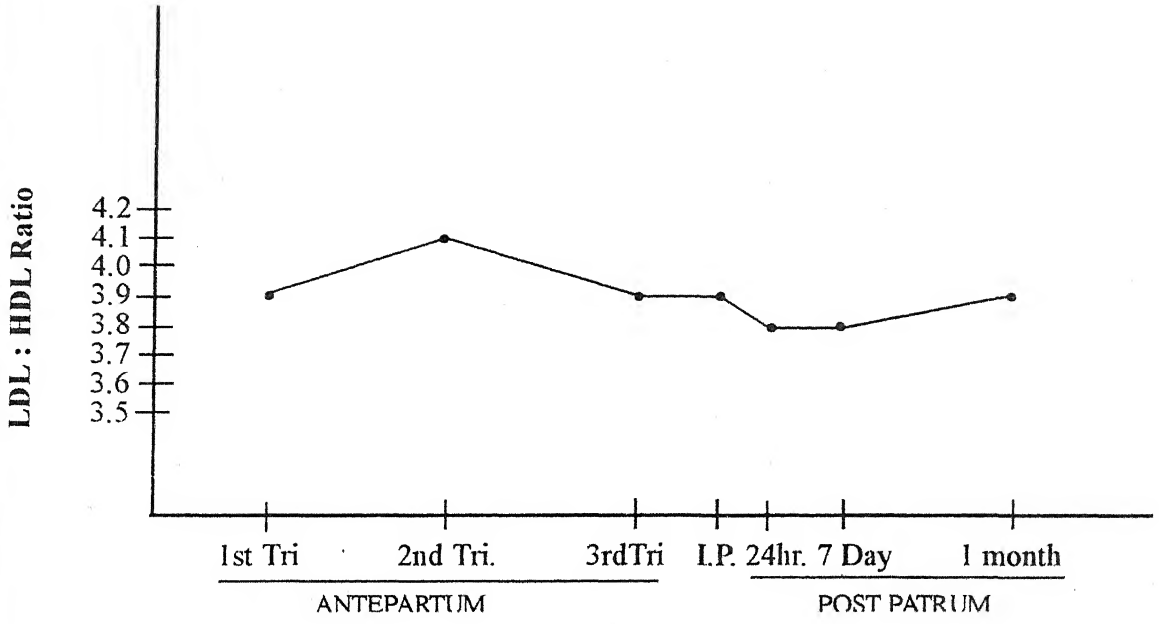


TABLE VI : Showing mean values of LDL/HDL ratio during antepartum, intrapartum and postpartum period (Mean±S.D.).

	Trimester			Intra-partum	Post partum		
	I	II	III		24hr	7 day	1month
LDL:HDL	3.83 ±0.56	4.18 ±0.98	3.96 ±0.51	3.98 ±0.82	3.87 ±0.82	3.83 ±0.63	3.95 ±0.61
n	32	38	46	56	47	32	28

Statistical Analysis

Groups compared	't' value	'p' value
I Vs II	2.00	7 0.05
I Vs III	0.001	7 0.05
I Vs IP	0.06	7 0.05
I Vs PP24h	0.63	7 0.05
I Vs PP7d	0.92	7 0.05
I Vs PP1m	0.23	7 0.05
II Vs III	1.9	7 0.05
II Vs IP	1.96	7 0.05
IP Vs PP24h	0.04	7 0.05
IP Vs PP7d	0.05	7 0.05
IP Vs PP1m	0.06	7 0.05

Statistical analysis shows though there was some difference in LDL : HDL ratio between antenatal, intrapartum and post partum periods. It was statistically insignificant.

GRAPH 07

Bar Diagram comparing serum lipoprotein levels in maternal & cord blood (placental end) at the time of delivery

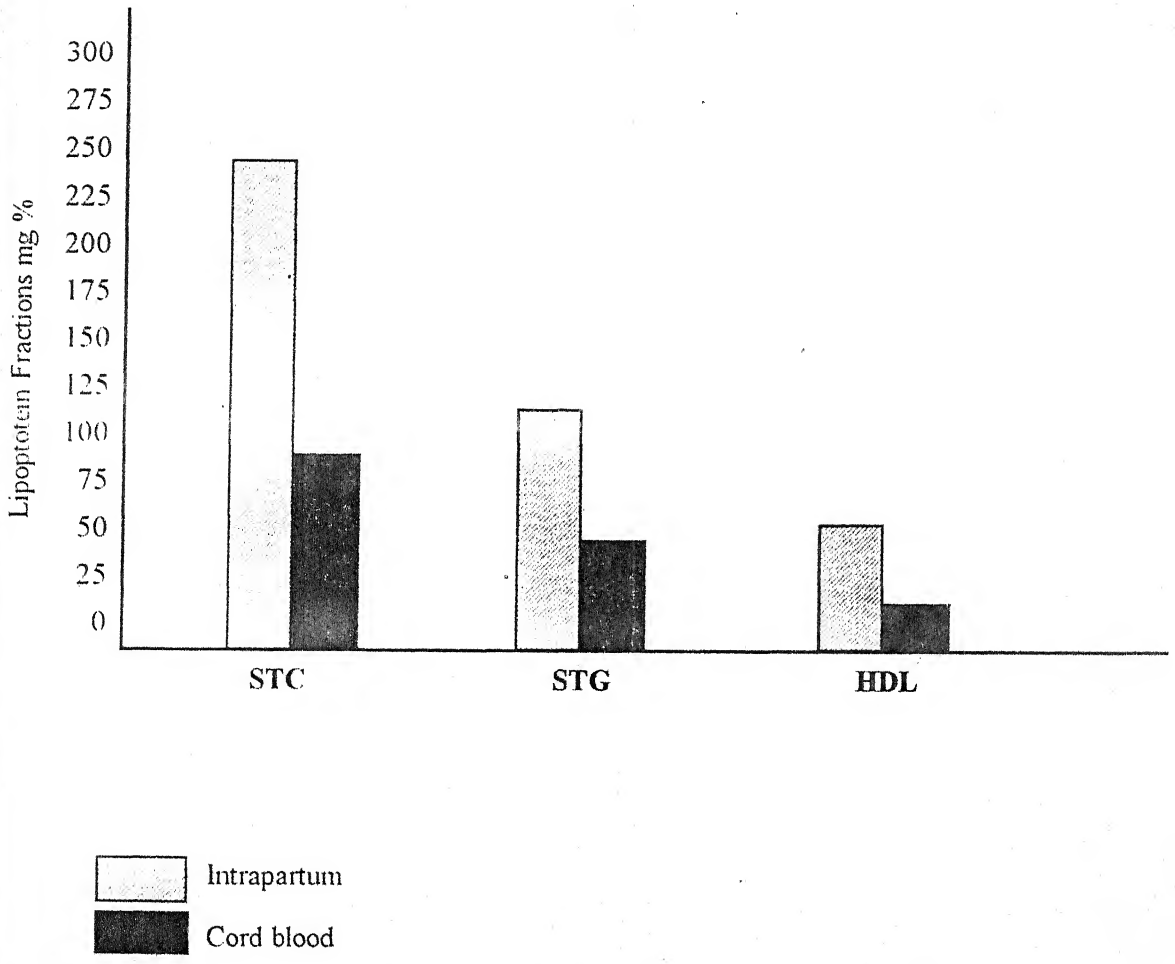


TABLE VII : Showing various lipoprotein fractions in umbilical cord blood of newborns taken from both placental and foetal end compared with intrapartum values of mother (Mean±S.D., mg/dl).

Lipo-proteins	Intrapartum (n=38)	Cord blood	
		Placental end (n=38)	Foetal end (n=21)
STC	242.60±22.03	94.76±19.18	92.50±10.40
STG	120.34±16.03	44.18±11.69	42.50± 5.90
HDL	46.00± 4.61	19.06± 2.07	18.97± 1.17
LDL	172.53±21.20	66.85±11.89	64.70± 7.56
VLDL	24.06± 3.31	8.83± 1.62	8.70± 1.20

Table VII shows that STC of 242.60±22.03 mg/dl at the intrapartum period in the maternal blood while in the umbilical cord blood of the newborns, STC was 94.76± 19.18 and 92.50±10.40 mg/dl of the placental and foetal ends of the cord respectively.

STG level in the maternal blood was 120.34±16.03 mg/dl while in the umbilical cord blood STG was 44.18± 11.69 mg/dl at placental end and 42.50±5.90 mg/dl at foetal end. Similarly HDL in the maternal blood was 46.00±4.61 mg/dl while in cord blood it was 19.06±2.07 and 18.97±1.17 mg/dl at placental and foetal end respectively.

Statistical Analysis

Groups compared	'p' values		
	STC	STG	HDL
IP Vs CBP	< 0.001	< 0.001	< 0.001
IP Vs CBF	< 0.001	< 0.001	< 0.001
CBP Vs CBF	> 0.05	> 0.05	> 0.05

Statistical analysis shows that lipoprotein fractions in the umbilical clord blood (placental and foetal ends) were significantly lower when compared with their maternal blood levels during labour.

TABLE VIII : Showing values of lipoproteins in lactating and non-lactating mothers at 7 days postpartum (Mean$\pm$S.D., mg/dl).

Lipo-proteins	Lactating (n=25)	Non-lactating (n=7)
STC	207.72$\pm$19.95	224.57$\pm$24.08
STG	100.68$\pm$10.89	112.14$\pm$18.31
HDL	38.96$\pm$ 3.07	40.57$\pm$ 2.81

Statistical Analysis

Comparison between lactating and nonlactating values.

	't' value	'p' value
STC	1.89	> 0.05
STG	1.67	> 0.05
HDL	0.46	> 0.05

Table VIII shows that at 7 days postpartum STC level was 207.72$\pm$19.95 mg/dl in lactating group while in nonlactating group it was higher(224.57$\pm$24.08 mg/dl).

STG in lactating mothers was 100.68±10.89 mg/dl while in nonlactating group it was 112.14±18.31 mg/dl. Similarly HDL was 38.96±3.07 mg/dl in lactating group and 40.57±2.81 mg/dl in non-lactating group.

Statistical analysis of lipoprotein levels at 7 days postpartum between lactating and non-lactating groups showed insignificant difference.

TABLE IX : Showing lipoprotein levels in lactating and nonlactating mothers at 1month post-partum (Mean±S.D., mg/dl).

Lipo-proteins	Lactating (n=19)	Nonlactating (n=7)
STC	198.31±20.02	224.85±26.67
STG	98.78±16.35	110.85±13.17
HDL	36.94± 5.18	38.14± 2.96

Statistical Analysis

Comparison between lactating and nonlactating groups.

Lipo-proteins	't' value	'p' value
STC	2.768	< 0.05
STG	1.754	> 0.05
HDL	0.58	> 0.05

Table IX also shows a similar trend in lipoprotein fractions at 1 month postpartum like 7 days postpartum values e.i. all the lipoprotein fractions were higher in the nonlactating group than that of lactating group.

Statistical analysis showed that the difference in STC at 1 month postpartum between lactating and nonlactating group was statistically significant while the difference in STG and HDL between these two groups was insignificant.

TABLE X : Lipoprotein changes in relation to mode of delivery during intrapartum and postpartum. (Mean±S.D., mg/dl).

Lipo-protein	Intra-partum	Post partum period		
		24 hrs	7 days	1 month
Group I				
STC	248.60 ±22.49	225.60 ±26.89	219.70 ±18.34	219.50 ±19.35
STG	122.76 ±16.69	109.15 ±11.42	107.44 ±14.84	110.00 ±12.81
HDL	44.44 ± 4.12	41.50 ± 3.95	41.00 ± 3.80	39.30 ± 4.16
Group II				
STC	240.46 ±21.24	221.50 ±18.22	214.77 ±20.26	202.42 ±23.45
STG	122.38 ±18.00	115.75 ±16.89	106.66 ±12.94	102.28 ±19.74
HDL	45.83 ± 5.83	42.90 ± 3.83	39.22 ± 2.92	36.00 ± 4.04
Group III				
stc	237.58 ±26.65	216.10 ±30.71	200.30 ±23.07	196.37 ±38.24
STG	112.75 ±13.79	106.40 ±11.74	96.10 ±11.90	96.75 ±15.56
HDL	44.66 ± 4.26	43.60 ± 3.43	39.90 ± 3.38	36.97 ± 2.80

Group I : Subjects who had vaginal delivery.

Group II : Subjects who had an emergency caesarean section

Group III : Subjects who underwent an elective caesarean section.

Table X shows that group I comprised of 26 subjects who had vaginal delivery. Lipoprotein fractions (STC, STG and HDL) showed maximum values at intrapartum period. It declined suddenly within 24 hours postpartum and further drop occurred during 7 days and 1 month postpartum.

Group II comprised of 13 subjects who had an emergency caesarean section. and group III comprised of 13 subjects who had undergone elective caesarean section. Both the groups II and III showed a similar trend in lipoprotein levels as that of group I i.e. maximum values at intrapartum period followed by a fall in the postpartum period as shown in table X.

Statistical Analysis

Comparison : Vaginal delivery Vs Emergency caesarean section.(Group I Vs II).

Lipoprotein	Period	't' value	'p' value
STC	Intrapartum	1.04	7 0.05
	PP24 h ours	0.45	7 0.05
	PP 7days	0.57	7 0.05
	PP 1 month	1.52	7 0.05
STG	Intrapartum	0.06	7 0.05
	PP 24 hours	1.29	7 0.05
	PP 7 days	0.11	7 0.05
	PP 1 month	0.94	7 0.05
HDL	Intrapartum	0.83	7 0.05
	PP 24 hours	0.94	7 0.05
	PP 7 days	1.07	7 0.05
	PP 1 month	1.52	7 0.05

Group I Vs III(Vag. delivery Vs Elective caesarean)

STC	Intrapartum	1.31	7 0.05
	PP 24 hours	0.83	7 0.05
	PP 7 days	1.91	7 0.05
	PP 1 month	1.64	7 0.05
STG	Intrapartum	1.80	7 0.05
	PP 24 hours	0.16	7 0.05
	PP 7 days	1.75	7 0.05
	PP 1 month	1.87	7 0.05
HDL	Intrapartum	0.05	7 0.05
	PP 24 hours	1.37	7 0.05
	PP 7 days	1.39	7 0.05
	PP 1 month	1.35	7 0.05

Analysis shows that the difference in lipoprotein changes between various groups were statistically insignificant.

TABLE XI : Lipoprotein levels in relation to outcome of pregnancy(live birth Vs still birth) during antepartum, intrapartum and postpartum periods. (Mean±S.D., mg/dl).

Lipo-protein	Trimester II	Trimester III	Intra-partum	Postpartum period 24hr	Postpartum period 7 days	Postpartum period 1 month
STC						
Group A	217.26 ±23.63	240.05 ±24.96	241.38 ±23.02	220.68 ±25.10	210.31 ±19.96	205.86 ±24.89
Group B	214.00 ±36.49	240.83 ±37.16	257.16 ±33.71	231.83 ±22.70	222.00 ± 5.29	207.40 ±29.89
STG						
Group A	99.05 ±13.98	118.30 ±17.40	119.38 ±16.80	110.02 ±12.80	102.96 ±13.01	102.86 ±15.08
Group B	95.00 ±20.66	121.83 ±21.97	132.50 ±20.62	113.66 ±17.23	104.75 ±13.88	103.40 ±13.44
HDL						
Group A	37.79 ± 5.08	44.13 ± 6.32	44.62 ± 7.34	42.56 ± 5.97	39.67 ± 4.30	37.52 ± 4.49
Group B	37.00 ± 4.35	42.83 ± 4.06	44.33 ± 1.20	40.50 ± 2.34	39.00 ± 2.94	36.20 ± 3.00

Group A : there were 52 subjects who delivered live baby.
Group B : There were 7 subjects who delivered dead baby (still birth).

Statistical Analysis

Comparison : Live births Vs still birth.

Periods	STC		STG		HDL	
	't'	'p'	't'	'p'	't'	'p'
Trimester II	0.06	70.05	0.46	70.05	0.04	70.05
III	0.06	70.05	0.09	70.05	0.32	70.05
Intrapartum	1.49	70.05	1.75	70.05	0.09	70.05
Postpartum 24hr	0.96	70.05	0.82	70.05	0.09	70.05
7 days	1.06	70.05	0.62	70.05	0.07	70.05
1 month	0.80	70.05	0.09	70.09	0.06	70.05

Table XI shows that there were 52 subjects in group A and 7 subjects in group B. STC levels in either groups do not show much difference during 2nd and 3rd trimester. The respective values were 217.26 ± 23.63 and 240.05 ± 24.96 mg/dl in group A while in group B it was 214.00 ± 36.49 and 240.83 ± 37.16 mg/dl in 2nd and 3rd trimester respectively. But during intrapartum period, postpartum 24 hours, 7 days the STC level in group B were higher when compared with that of group A as shown in table XI.

Serum triglycerides levels were also higher in group B than that of group A during 3rd trimester, intrapartum, 24 hours, 7 days and 1 month postpartum period as shown in table XI.

Statistical analysis showed that the difference in lipoprotein levels (STC, STG, HDL) between live birth Vs still birth(Group A Vs B) were statistically insignificant.

Note : All the 7 subjects of group B had normal antenatal period. Four out of 7 still births were vaginal delivery and 3 were emergency caesarean section. Birth weight of all the babies was 72500 gms.

TABLE XII : Lipoprotein levels in relation to parity during antepartum, intrapartum and postpartum periods (Mean±S.D., mg/dl).

Lipoprotein	Trimester I	Trimester II	Trimester III	Intrapartum	Post partum 24 hr	Post partum 7 days	Post partum 1month
STC							
Primi	172.20 ±15.40	217.20 ±21.10	235.80 ±19.50	238.80 ±20.41	218.60 ±16.40	214.40 ±19.30	197.60 ±16.21
Multi	162.10 ±22.61	208.20 ±26.20	234.80 ±20.41	245.60 ±25.43	223.90 ±24.70	210.04 ±21.30	210.15 ±25.64
STG							
Primi	91.81 ±21.60	98.76 ±23.60	111.93 ±20.41	117.66 ±21.44	107.43 ±17.40	105.27 ±18.41	97.77 ±18.31
Multi	92.76 ±24.60	94.76 ±18.44	118.25 ±27.41	124.08 ±26.31	112.06 ±23.61	102.09 ±22.20	105.40 ±25.10
HDL							
Primi	32.07 ± 3.60	38.30 ± 2.10	43.80 ± 3.61	44.80 ± 4.12	42.25 ± 3.80	40.00 ± 2.74	36.30 ± 2.84
Multi	30.64 ± 5.40	35.92 ± 4.9	42.61 ± 2.34	44.45 ± 3.65	42.32 ± 2.60	39.38 ± 3.16	37.73 ± 4.34

Primi = Primigravidae consisting 22 subjects.

Multi = Multigravidae consisting 38 subjects.

Statistical Analysis

Comparison : Primi Vs Multi.

Period	STC 't'	STC 'p'	STG 't'	STG 'p'	HDL 't'	HDL 'p'
Trimester I	1.91	70.05	0.60	70.05	1.10	70.05
II	1.92	70.05	1.10	70.05	1.30	70.05
III	0.06	70.05	1.60	70.05	0.60	70.05
Intrapartum	1.70	70.05	1.12	70.05	0.02	70.05
Postpartum : 24 hrs	0.90	70.05	0.90	70.05	0.01	70.05
7 days	1.10	70.05	0.31	70.05	0.12	70.05
1 month	0.60	70.05	1.20	70.05	0.32	70.05

Table XII shows that STC levels in 1st, 2nd and 3rd trimester in the primigravidae (172.20±15.40, 217.20± 21.10 and 235.80±19.50 mg/dl respectively) were numerically higher than the respective values in multigravidae i.e. 162.10±22.61, 208.20±26.20 and 234.80±20.41 mg/dl respectively. While in intrapartum, 24 hours and 1 month post partum the STC level in primigravidae were 238.80±20.41, 218.60±16.40 and 197.60±16.20 mg/dl respectively and were lower than the respective values in multigravidae i.e. 245.60±25.43, 223.90±24.70 and 210.15±25.64 mg/dl respectively.

HDL values also showed a numerically higher values in primigravidae during 1st, 2nd and 3rd trimester as shown in table XII.

Statistical analysis of the difference in lipoprotein fractions between primigravidae and multigravidae during antepartum, intrapartum and postpartum periods was not significant ($p > 0.05$).

D I S C U S S I O N

D I S C U S S I O N

Various studies have shown that in normal pregnancy there is an increase in serum total cholesterol and serum triglycerides which progresses steadily till term and then abruptly falls after delivery (Boyd, 1934; Dieckman and Wegner, 1934; and Watson, 1957).

With this back ground the present study was conducted to know the lipoprotein pattern in normal pregnancy and puerperium so as to know the lipoprotein changes in relation to parity, lactational status, mode of delivery (vaginal delivery, caesarean section) and pregnancy outcome (live birth, still birth).

Of the 104 subjects enrolled in this study, only 60 subjects were considered for final analysis. The rest being dropped at different stages of study due to lack of sufficient follow up or because of they developed complications like pre-eclampsia, eclampsia, gestational diabetes etc during follow up.

The basal level of plasma lipids in normal subjects are as follows :

STC	: $\angle$ 200 mg/dl	LDL	: 50-130 mg/dl
STG	: $\angle$ 160 mg/dl	VLDL	: $\angle$ 32 mg/dl
HDL	: 30-90 mg/dl		

(Annal. Int. Med., 1993; 119(7) Part 1).

SERUM TOTAL CHOLESTEROL (STC)

In this study, STC was 168.78±33.01 mg/dl in first trimester which increased through 2nd and 3rd trimester (217.40±25.10 and 240.15±22.11 mg/dl) respectively) to reach a peak level of 243.07±25.72 mg/dl at intrapartum period. The STC level then declined abruptly to 222.10± 26.52 mg/dl within 24 hours post partum. Further decrease occurred 7 days (211.40±20.44 mg/dl) and 1 month (206.14± 26.12 mg/dl) postpartum. The STC level at 1 month post-partum (206.14±26.12 mg/dl) was still higher than the value of 1st trimester (168.78±33.01 mg/dl).

This trend in lipoprotein changes in pregnancy and puerperium was generally in accordance with the observations previously published by Boyd and Oliver (1955), Mullick and Bagga (1964), Konttinen et al (1964), Pontis et al(1978), Udoh and Etam et al (1994) and Chiang and Yang et al (1995).

Previous study from this department (Arora and Garg K et al, 1989) also showed a similar trend in lipoprotein pattern in pregnancy and puerperium. But the respective values observed by them were lower than the present study. In their study the 1st trimester STC level was 149± 26.40 mg/dl and intrapartum level was 230.28±32.10 mg/dl.

Arora and Vinita et al (1987) showed STC level of as 166.17±24.97 mg/dl in 1st trimester which reached a peak of 263.44±39.80 mg/dl during labour then decreasing in the postpartum period reaching 190.50±36.94 mg/dl at 1 month postpartum.

SERUM TRIGLYCERIDES (STG)

In this study, STG level was 95.41±10.25 mg/dl in 1st trimester which progressively increased in 2nd and 3rd trimester reaching a peak value of 120.78±17.23 mg/dl at intrapartum period. The STG level then declined sharply to 110.48±13.09 mg/dl within 24 hours of delivery. The STG level further declined in puerperium reaching 102.96± 15.70 mg/dl at 1month postpartum. The level of STG at 1 month postpartum was significantly higher than that of 1st trimester levels. The STG level at 3rd trimester and intrapartum when compared with that of 1st trimester were statisticallt highly significant. These findings were in accordance with previous observations (Sitadevi and Patrudu et al, 1981; Chiang & Yang et al, 1995 and Udoh et al,1994).

Hachy et al (1994) reported a 200-400% increase in serum triglycerides during normal pregnancy by term. In our study the increase in STG level from 1st trimester to intrapartum was only 26.59%.

The rising maternal plasma titre of oestorgen appear to be the principal hormonal factor responsible for the enhanced endogenous synthesis of triglycerides.

HDL CHOLESTEROL (HDL-c)

In this study, the HDL-c level was 31.01±3.61 mg/dl in 1st trimester which gradually increased as the advancement of pregnancy reaching a peak value of 44.58±7.33 mg/dl during labour. The HDL-c level then decreased in the postpartum period reaching 37.28±4.48 mg/dl at 1 month postpartum. The level of HDL-c at 2nd and 3rd trimester and

intrapartum were highly significant statistically when compared with that of first trimester ($p < 0.001$). The HDL-c level at 1 month postpartum was higher than that of 1st trimester and the difference was statistically significant.

As we know that oestrogens increase HDL-c level, these findings can be correlated with the level of oestrogen during pregnancy and in postpartum period.

The above findings are in accordance with previous observations by Chiang and Yang et al (1995).

Udoh and Ndem et al (1994) reported 35±10% increase in HDL-c from 3 to 9 months of pregnancy. In this study, the mean increase in HDL-c from 1st trimester to intrapartum period was 43.76%.

LDL CHOLESTEROL (LDL-c)

The present study showed that LDL-c was 118.84± 30.86 mg/dl at 1st trimester which showed a statistically significant increase to 159.10±20.12 mg/dl at 2nd trimester. The value reached at peak to 172.55±20.79 mg/dl at intrapartum period. The rise was highly significant ($p < 0.001$) when compared with that of 1st trimester. The LDL-c then showed a significant ($p < 0.001$) drop within 24 hours of delivery. Further fall occurred during puerperium reaching a value of 148.35±22.30 mg/dl at 1 month postpartum. The value at 1 month postpartum was still higher than that of 1st trimester and the difference was statistically significant.

Previous study in this department (Arora & Garg K et al, 1989) showed comparatively low values of LDL-c during pregnancy and puerperium. In that study LDL-c was 93.70±30.95 mg/dl at 1st trimester, 119.50±25.60 mg/dl during intrapartum and 92.85±24.46 mg/dl at 1 month postpartum, but the changing trend was similar to present study. The gross disparity in LDL-c values between previous study and present study can be explained that LDL-c is a derived fraction of STC and depends on the value of STC, HDL and VLDL. In the present study STC levels were relatively higher than the previous study in each trimester and puerperium. Moreover, the HDL-c and VLDL values were comparatively lower than the previous study, so naturally the derived LDL-c value remained high.

VLDL

VLDL values are derived from STG values (VLDL=STG/5). So the pattern of VLDL in pregnancy and puerperium showed a parallel relationship as that of serum triglycerides. At 1st trimester VLDL was 19.08±2.02 mg/dl which raised significantly ($p < 0.05$) to 20.20±2.66 mg/dl in 2nd trimester. The value reached at 24.15±3.42 mg/dl at intrapartum period. The rise was highly significant ($p < 0.001$) when compared with 1st trimester level. The LDL level then decreased significantly ($p < 0.001$) within 24 hours of delivery. The VLDL level at 1 month postpartum was higher than that of 1st trimester level, but the difference was statistically insignificant.

Previous study (Arora and Garg K et al, 1989) reported a similar trend in VLDL changes in pregnancy and puerperium.

LDL : HDL RATIO

As shown in table VI, the LDL:HDL ratio remained more or less stationary throughout pregnancy, intrapartum and puerperium, with no significant change ($p > 0.05$) between these periods. Thus it may inferred that the rise in LDL-c (as also STC) tallied with the rise in HDL-c and hence the ratio remained rather static.

Mazurkiewicz and Watts et al (1994), in their study of serum lipids, lipoproteins and apoproteins in pregnant non-diabetic patients, showed that total cholesterol : HDL-c ratio was not significantly different in normal pregnancy.

CORD BLOOD

Lipoprotein fractions in the umbilical cord blood showed a low value when compared with the maternal values in intrapartum period. This was true with the cord blood samples taken from the placental and foetal end of the cord.

STC at intrapartum was 242.60 ± 22.03 mg/dl while the cord blood (placental end) showed 94.76 ± 19.18 mg/dl. The difference being statistically highly significant ($p < 0.001$). Cord blood from the foetal end showed 92.50 ± 10.40 mg/dl when the STC level at placental and foetal ends were compared, no statistically significant difference was found.

STG level at intrapartum period was 120.34 ± 16.03 mg/dl while in cord blood it was 44.18 ± 11.69 mg/dl

(placental end) and 42.50±5.90 mg/dl(feotal end). The difference between intrapartum and cord blood was highly significant (p <0.001), while the difference in STG levels between placental and foetal ends samples were statistically insignificant.

Intrapartum HDL levels was 46.00±4.61 mg/dl in maternal blood while in cord blood it was 19.06±2.07 mg/dl (placental end) and 18.97±1.17 mg/dl (foetal end). The difference in HDL level between maternal blood and cord blood was highly significant (p <0.001).

VLDL and LDL also showed a similar significantly low values in umbilical cord blood in comparison to the maternal blood during labour (Table VII).

Normal values of STC in cord blood ranged 45-98 mg/dl and STG from 10-98 mg/dl (Cloherty Stark, Manual of Neonatology).

Pontis et al (1978) reported that the average values of lipoprotein in umbilical cord blood were far below that of maternal blood. The difference that exists between mother and baby in this respect varies from case to case and values prevailing in one seems to be entirely independent of those in others.

EFFECT OF LACTATION

On comparing the lipoprotein fractions (STC, STG, HDL) in lactating and non-lactating mothers, all the fractions showed a numerically high value in non-lactating

group at postpartum 7 days and 1 month. But on statistical analysis the difference in lipoprotein between two groups were found to be insignificant ($p > 0.05$) at 7th day post-partum.

At 1 month postpartum, the STC in non-lactating group was 224.85 ± 26.67 mg/dl while in lactating group the STC was 198.31 ± 20.02 mg/dl. The difference was significant ($p < 0.05$). At 1 month postpartum, STG and HDL showed no significant difference between the two groups (Tables VIII and IX).

Darmandy and Postle et al (1982) reported that there was a considerable variation in the time required for the hyperlipidemia of pregnancy to decline after delivery. Lactation appeared to be an important factor in this variation, women who bottle fed their infants maintained an elevated STG level for three times longer than those who breast fed their infants.

According to Boyd (1935) the lipid concentration of blood plasma was found to decline consistently after delivery in all cases where normal lactation occurred.

Arora and Vinita et al (1987) found that STC level in normal lactation when compared with insufficient lactation or no lactation were highly significant.

EFFECT OF MODE OF DELIVERY

Arora and Neeta et al (1993) studied the changes in lipoprotein profile in normal pregnancy and artificial

termination of pregnancy (Elective/Emergency LSCS). They observed a rising trend in lipoprotein profile with peak during labour followed by a fall in the postpartum period both in normal as well as artificial termination of pregnancy.

In our study, subjects were divided into 3 groups. Group I(vaginal delivery), group II (Emergency caesarean) and group III (Elective caesarean). All the three groups showed similar trend in lipoprotein pattern i.e. maximum values at intrapartum period and a sharp decline in lipoprotein fractions after delivery irrespective of mode of delivery. Statistical analysis between three groups showed no significant difference in lipoprotein fractions.

Arora and Juhi Arora et al (1994) also found similar observations. They concluded that the process of labour does not bring about any significant changes in the lipoprotein profile.

PREGNANCY OUTCOME

Present study on lipoprotein levels in relation to pregnancy outcome (Live birth and still births) showed that STC levels in either groups (Group A - live birth and Group B - still birth) did not show much difference during 2nd and 3rd trimester. But during intrapartum, 24 hours, and 7 days postpartum, the STC levels in group B were higher as compared to that of group A (Table XI).

STG levels were also higher in group B than that of group A during 3rd trimester, intrapartum, 24 hours,

7 days and 1 month postpartum. Statistica analysis showed that the difference in lipoprotein levels (STC, STG & HDL) between group A and B were not significant ($p > 0.05$). Since the sample size in group B was very small (n=7) it was improper to come to any conclusion with these observations.

EFFECT OF PARITY

Lipoprotein changes in relation to parity(primi, multi) were compared and given in table XII. Results showed that the STC levels were higher in the primigraviade during 1st, 2nd and 3rd trimesters in comparison to multigravidae, but the difference was statistically insignificant. During intrapartum, 24 hour and 1 month postpartum the STC values were higher in multigravidae but here also it was statistically insignificant.

Comparison of STG levels between primigravidae and multigravidae also failed to show any significant difference between either groups.

HDL levels showed a slightly high values in primigravidae during 1st, 2nd and 3rd trimester in comparison to multigravidae but the difference was statistically insignificant.

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

The present study was conducted to know the lipoprotein changes during normal pregnancy and puerperium and also to know the changes in lipoprotein pattern in relation to parity, lactation, mode of delivery and outcome of pregnancy (Live birth and still birth).

Sixty cases (22 primi and 38 multi in the age from 17 to 36 years) of normal pregnancy were followed up through antepartum, intrapartum and postpartum periods.

Lipoprotein levels (STC, STG, HDL, LDL and VLDL) were estimated during 1st, 2nd and 3rd trimester, intrapartum, 24 hour, 7 days and 1 month postpartum. Lipoprotein levels in umbilical cord blood of newborns(Both from placental and foetal ends) were also noted.

STC levels at 1st trimester was 168.78±33.01 mg/dl which showed a significant increase ($p < 0.001$) by second trimester. Further increase occurred in 3rd trimester reaching a peak value of 243.07±23.33 mg/dl at intrapartum period. The STC level then declined significantly($p < 0.001$) by 24 hours of delivery to 222.10±26.52 mg/dl. Further decrease occurred in postpartum 7 days and by 1 month postpartum STC level was 206.14±26.12 mg/dl.

STG raised from 95.41±10.25 mg/dl at 1st trimester to 98.72±13.73 mg/dl at 2nd trimester. Further increase occurred in 3rd trimester reaching the maximum value of

120.78±17.23 mg/dl at intrapartum period. STG level then decreased significantly ($p < 0.001$) by 24 hours of delivery (110.48±13.09 mg/dl). Further decrease occurred during 7 days postpartum and 1 month postpartum.

HDL cholesterol raised from 31.01±3.61 mg/dl at 1st trimester to 37.72±5.19 mg/dl at 2nd trimester reaching a peak level of 44.58±7.33 mg/dl at intrapartum period. The HDL-c then declined significantly ($p < 0.05$) at 24 hour post partum. Further decrease occurred at 7 days postpartum and at 1 month postpartum these values reached to 37.28±4.48 mg/dl.

VLDL and LDL cholesterol also showed a similar trend i.e. their levels increased progressively with the advancement of pregnancy reaching the peak value at intrapartum period and then declined significantly at 24 hours postpartum and further decrease occurred during postpartum 7 days and 1 month.

The LDL:HDL ratio showed no significant changes during various trimesters, intrapartum and postpartum periods.

On comparing the various lipoprotein fractions in the umbilical cord blood of newborn (Both placental and foetal ends of the cord) with that of maternal blood during labour, it was found that the level of various lipoprotein fractions in the umbilical cord blood were far lower ($p < 0.001$) than that of maternal blood. Although the level of lipoprotein fractions in the sample taken from the

foetal end of umbilical cord showed a slightly lower value than that taken from the placental end, the difference was statistically insignificant ($p > 0.05$).

While noting the effect of lactation in lipoprotein profile, we found that at postpartum 7 days, the lipoprotein fractions showed a numerically high value in the non-lactating group as compared to lactating group, but this difference was statistically insignificant ($p > 0.05$). At 1 month postpartum, the STC in non-lactating group was 224.85 ± 26.67 mg/dl while in lactating group it was 198.31 ± 20.02 mg/dl, the difference was statistically significant ($p < 0.05$). STG and HDL levels were also higher in non-lactating group as compared to lactating group at 1 month postpartum, though the difference was insignificant($p > 0.05$).

To know the effect of mode of delivery on lipoprotein fractions, the subjects were divided into three groups (Group I - vaginal delivery, Group II - emergency caesarean section and group III - Elective caesarean section). It was found that the decline in lipoprotein fractions following delivery were not related to the mode of delivery i.e. lipoprotein fractions showed a similar decline in all the three groups after delivery.

In the present study, 7 subjects who had an otherwise normal pregnancy, delivered dead babies (birth weight > 2500 gms in all cases). When the lipoprotein pattern of mother who had live birth (Group A) were compared with that of mothers who had still birth (Group B) it was found that

the difference between these two groups was not statistically significant.

To note the effect of parity on lipoprotein profile, the cases were divided into 2 groups i.e. primigravidae consisting 22 subjects and multigravidae consisting 38 subjects. Lipoprotein fractions showed similar changes in either groups during antepartum, intrapartum and postpartum periods. The intergroup difference was statistically insignificant.

To conclude, there was a statsitically significant rise in all lipoprotein fractions(STC, STG, HDL-c, LDL-c and VLDL) with the advancement of pregnancy. The highest values were recorded in the intrapartum phase, followed by a significant fall in the early postpartum phase. These changes were not affected by factors like parity, lactatory status or mode of delivery. It may be presumed that the observed changes are normal physiological phenomenon.

B I B L I O G R A P H Y

BIBLIOGRAPHY

1. Arora RC, Arora S, and Vinita. Evaluation of effect of dietary fat on serum total cholesterol level during antepartum, intrapartum and postpartum periods of pregnancy and in cord blood of neonates, 1987(Thesis for M.S. Obst. & Gynaecology).
2. Arora S and Malik I. Study of antepartum and post-partum serum total cholesterol level in normal pregnancy and toxaemia of pregnancy, 1986 (Thesis for M.S., Obst. & Gynaecology).
3. Arora RC, Arora S, Garg K, Agarwal N. Study of changes of lipid lipoprotein profile in normal pregnancy and toxaemia of pregnancy during antepartum, intrapartum and postpartum periods and umbilical cord blood of their newborns. 1989 (Thesis for M.S. Obst & Gynaec.).
4. Annals of Internal Medicine, Vol 119(7) Part 1. Lab values of lipoproteins.
5. Adlersberg D. Hormonal influence on the serum lipids. Am J Med, 1957; 23 : 769.
6. BacMeister and Haverz. Deutsch Med Wehschr, 1914; Vol 4 : 385.
7. Bacqueral A and Rodies A (1945). Untersuchunger Liber die Zuzemmensetzung des blutes EV Langen.
8. Bhattacharya V and Amma MKP. Lipid metabolism in pregnancy. Indian J Obst & Gynaec, 1969; P. 682.
9. Boyd EM. J Clin Invest, 1934; 13 : 347.

10. Boyd EM. Canadian Med Assoc J, 1935.

11. Boyd EM. Diurnal variation in plasma lipids. J Biol Chem, 1935; 11 : 61.

12. Boyd EM and Kingston CM. Blood lipids in pre-eclampsia. Am J Obst & Gynaec, 1936; 32 : 937.

13. Boyd EM and Bochesler NY. Blood lipids in the puerperium. Am J Obst & Gynaec, 1935; 29 : 797.

14. Boyd EM and Wilson KM. The exchange of lipids in the umbilical circulation at birth. J Clin Invest, 1935; 14 : 7.

15. Chaturvedi D, Tandon S, Singh KN. Serum cholesterol in pregnancy. Indian J Obst & Gynaec, 1978; XXVIII (4) : 502-505.

16. Chanfford A, Laroche G and Grigguat A. Obstetrique, Sciences 1911; 4 : 481.

17. Chiang AN, Yang ML, Hung JH and Chou P. Life Sciences, 1995; 56(26) : 2367-75.

18. Darmandy JM, Postle AD. Lipid metabolism in pregnancy. Brit J Obst & Gynaec, 1982; 89(3) : 211-15.

19. De Alvarez RR and Bratvold GE. Serum lipid in pre-eclampsia and eclampsia. Am J Obst & Gynaec, 1961; 81 : 1140.

20. Dieckman WJ, Chicogo, Illinosis : Comparative studies of the blood in the nonconvulsive toxaemia of pregnancy. Am J Obst & Gynaec, 1933; 26 : 543.

21. Dieckman WJ and Wegner CR. Studies of the blood in normal pregnancy. Arch Inter Med, 1934; 53 : 540.

22. Erkkola R, Viikari J, Irjala K. One year follow up of lipoprotein metabolism after pregnancy. Biol Research in Pregnancy & Perinatology, 1986; 7 (2) : 47-51.

23. Fredrickson DS. A physiology guide to hyperlipidemia. Med Concept of cardiovascular disease, 1972; 41 : 31.

24. Green JC. Serum cholesterol in pregnancy. Am J Obst & Gynaec, 1966; 95 : 387.

25. Hamosh M, Clary TR, Cherwick SS and Scow RO. Lipoprotein lipase activity in adipose tissue and mammary tissue and plasma triglycerides in pregnant and suckling rats. Biochem Biophys Acta, 1970; 210 : 473-482.

26. Hachey DL, Benefits and risks of modifying maternal fat intake in pregnancy and lactation. Am J Clinic Nutrition, 59 (2 suppl) : 4545-4635.

27. Hermann and Neumann. Biochem Ztachr, 1912; 43 : 47.

28. Jailer JW and Laysam D. Hormonal changes during pregnancy. The Surg Clin North Amer, 1957; 37 : 341.

29. Kalkhoff RK, Kissebah AM and Kim NJ. Carbohydrate and lipid metabolism during normal pregnancy. Seminars in perinatology, 1978; 2 (4) : 291-307.

30. Knopp RH, Humphrey J and Irvine S. Biphasic metabolic control of hypertriglyceridemia in pregnancy. Clin Research, 126 : 4.

31. Konttinen A, Pyorala T and Carpen E. Serum lipid pattern in normal pregnancy and pre-eclampsia. J Obst & Gynaec Brit Comm, 1964; 71 : 453.

32. Kilby MD, Neery RH, Game FL et al. Foetal and maternal lipoprotein metabolism in human pregnancy. Clin Science (Colch), 1995; 88 (3) : 311-18.

33. Lall I and Sinha A. Study of antepartum and post-partum serum cholesterol level in normal and toxaemia of pregnancy. Indian J Obst & Gynaec, 1983; 33: 630.

34. Mullick S and Bagga OP. Serum lipid studies in pregnancy. Am J Obst & Gynaec, 1964; 89 : 766.

35. Majurkiewicz JC, Watts GF, Salvin BM et al. Serum lipid lipoprotein and apolipoprotein in pregnant non-diabetic patients. J Clin Path 1994;47(8) : 728-731.

36. Nelson GH, Zuspan PP and Mulligan LT. Defects of lipid metabolism in toxaemia of pregnancy. Am J Obst & Gynaec, 1966; 94 : 310.

37. Neumann J and Hermann E. Wein Klin Wetnschr, 1911; 24 : 441.

38. Oliver MF and Boyd GS. Plasma lipid and serum lipo-protein patterns during pregnancy and puerperium. Clin Sci, 1955; 14 : 15.

39. Ortega RM, Gasper MJ and Cantero M. Influence of maternal serum lipids and maternal diet during the 3rd trimester of pregnancy on umbilical cord blood lipids in the population of Spanish newborns. Intr J Vitamin and Nutri Research, 1996; 66(3):250-57.

40. Peters JP, Lleinmann M and Man EB. The lipids of serum in pregnancy. J Clin Invest, 1957; 30 : 388.

41. Pontis AV, Gupta SM and Purandare BN. Antepartum and postpartum cholesterol and lipoprotein levels in normal parturating women. Indian J Obst & Gynaec, 1978; 28 : 349.

42. Plass ED and Tempkins A. J Biol Chem, 1923; 56:309.

43. Schwarz PH, Soule SD and Dunie B. Blood lipids in pregnancy. Am J Obst & Gynaec, 1940; 39 : 203.

44. Sitadevi C, Patrudu MB. Longidutinal study of serum lipids and lipoproteins in normal pregnancy and puerperium. Trop & Geog Med, 1981; 33(3): 219-23.

45. Tyler and Under Hill FP. The influence of pregnancy upon the lipids of the blood. J Biol Chem, 1925; 66 : 1.

46. Udoh AE, Ndem ED, Itam EH, Odigwe CO. Serum cholesterol profile of some Nigerian pregnant women. Acta Medica, Hungwrea, 1994; 50(1-2) : 75-81.

47. Virchow R. Virchow's archeol Path Med, 1847; 44.

48. Watson WC. Serum lipids in pregnancy and puerperium. Clin Sci, 1955; 16 : 475.

M A S T E R C H A R T

MASTER CHART

A. GENERAL CHARACTERISTICS

Sl. No.	Name	Age (yrs)	Gravida	S.eco status	Diet	Mode of delivery	Outcome	Lactation
1.	Hasmukhi	25	1	M	N.V.	Elec.CS	Live	Lact.
2.	Diljit	30	4	M	N.V.	Em.CS	Dead	N.Lact.
3.	Parwati	25	2	L	..Veg.	V.D.	Live	Lact.
4.	Malti	24	2	M	Veg.	-	-	-
5.	Leela	25	2	M	Veg.	V.D.	Dead	Lact
6.	R.Kumari	25	2	L	Veg.	V.D.	Live	-
7.	Bindwasa	22	1	L	N.V.	Elec.CS	Live	Lact.
8.	Anita	23	1	M	Veg.	""	Live	Lact.
9.	Rashmi	21	1	M	Veg.	""	Live	Lact.
10.	Reena	25	2	L	N.V.	V.G.	Live	-
11.	Shagun	30	6	L	N.V.	V.D.	Live	Lact.
12.	Shashi	22	2	M	Veg.	V.D.	Live	Lact.
13.	Sona bai	24	3	L	N.V.	Elec.CS	Live	N.Lact.
14.	Kunti	30	4	L	N.V.	Em.CS	Live	""
15.	Malti	26	2	L	Veg.	Elec.CS	Live	Lact.
16.	Usha	22	2	M	Veg.	""	Live	Lact.
17.	Vidya	32	3	L	Veg.	Em.CS	Live	Lact.
18.	Vandana	30	1	M	N.V.	Elec.CS	Live	Lact.
19.	Kapoori	20	1	L	N.V.	V.D.	Live	-
20.	Rekha	22	1	M	Veg.	V.D.	Live	Lact.
21.	Meena	25	2	L	Veg.	V.D.	Live	-
22.	Meena	22	2	M	Veg.	Em.CS	Live	Lact.
23.	Kishori	36	5	L	N.V.	V.D.	Live	Lact.
24.	Meera	21	1	L	Veg.	V.D.	Live	-
25.	Devmani	24	2	M	Veg.	V.D.	Dead	N.Lact.
26.	Maya	18	1	L	Veg.	Em.CS	Live	Lact.
27.	Suman	23	3	L	N.V.	V.D.	Live	N.Lact.
28.	Vinita	23	1	M	Veg.	V.D.	Live	-
29.	Sheela	28	5	M	Veg.	Elect.CS	Live	Lact.
30.	Sarju	30	4	L	N.V.	V.D.	Live	-
31.	Uma	25	3	L	Veg.	Em.CS	Live	-
32.	Lakshmi	30	4	M	Veg.	Elect CS	Live	Lact.
33.	Meena	26	2	L	Veg.	Em.CS	Live	-

Cont....

34.	Rekha	21	1	M	Veg.	V.D.	Live	-
35.	Nazba	24	5	M	N.V.	V.D.	Live	Lact.
36.	R.Devi	22	2	L	Veg.	V.D.	Live	-
37.	Rekha	17	1	L	Veg.	V.D.	Live	-
38.	Urmila	25	2	L	Veg.	V.D.	Dead	N.Lact.
39.	Indira	25	2	L	N.V.	V.D.	Live	-
40.	Girja	25	1	M	N.V.	V.D.	Live	-
41.	Sunita	20	1	M	N.V.	V.D.	Live	N.Lact.
42.	Manorama	26	3	M	N.V.	V.D.	Live	-
43.	Geeta	22	1	L	Veg.	Em.CS	Live	-
44.	Mithla	18	1	L	N.V.	V.D.	Live	Lact.
45.	Urmila	25	3	L	Veg.	V.D.	Live	Lact.
46.	Usha	25	2	L	N.V.	V.D.	Dead	-
47.	Rajeshwari	25	4	M	Veg.	Elect.CS	Live	Lact.
48.	Shakuntala	20	1	M	N.V.	Em.CS	Dead	-
49.	Pinky	19	3	L	N.V.	V.D.	Live	-
50.	Malti	22	1	M	N.V.	Em.CS	Live	N.Lact.
51.	Meena	22	2	M	Veg.	Em.CS	Live	Lact.
52.	Mini	22	1	L	Veg.	Elect.CS	Live	-
53.	Munni	28	3	L	Veg.	V.D.	Live	Lact.
54.	Shanti	22	1	M	Veg.	Em.CS	Live	Lact.
55.	Khilan	32	4	M	N.V.	V.D.	Live	Lact.
56.	Ramkali	20	1	M	Veg.	Em.CS	Live	N.Lact.
57.	Seema	20	1	L	Veg.	V.D.	Live	-
58.	Santoshi	30	3	L	Veg.	Em.CS	Live	Lact.
59.	Keshkali	25	2	L	Veg.	Em.CS	Dead	Lact.
60.	Sarwari	24	4	M	N.V.	V.D.	Live	Lact.

M = Middle Socio-economic status.
L = Low socio-economic status,
Veg. = Vegetarian
N.V. = Non-vegetarian
V.D. = Vaginal Delivery
Em.CS= Emergency Caesarean section.
Elec.CS = Elective caesarean section.
Lact. = Lactating
N.Lact. = Non-lactating.

B. VALUES OF VARIOUS LIPOPROTEIN FRACTIONS

1. Serum Total Cholesterol (STC) (mg/dl).

Sl. No.	Trimester I	Trimester II	Trimester III	Intra-partum	Post partum 24h	Post partum 7d	Post partum 1m	CBP	CBF
1.		202	--	214	194	185	176	-	-
2.	-	-	200	-	188	185	180	-	-
3.	168	215	-	230	-	212	-	78	-
4.	167	218	240	-	-	-	-	-	-
5.	161	196	268	268	246	228	190	120	-
6.	130	-	200	-	190	-	-	-	-
7.	129	-	210	-	186	-	180	-	-
8.	130	214	222	235	210	195	181	94	-
9.	146	195	200	220	-	185	-	-	-
10.	220	225	275	280	-	-	250	-	-
11.	216	185	271	285	272	256	235	-	109
12.	-	-	-	208	200	-	-	-	-
13.	-	-	-	228	228	225	-	-	-
14.	198	200	255	260	240	252	235	94	-
15.	200	217	-	236	-	195	-	112	107
16.	138	-	-	198	-	185	170	-	-
17.	192	228	240	253	236	224	214	103	95
18.	-	218	-	228	-	196	-	86	-
19.	-	-	-	216	191	-	-	83	-
20.	232	250	255	255	246	226	216	112	-
21.	200	218	230	232	-	-	-	103	-
22.	-	-	-	215	192	190	-	-	-
23.	125	209	215	223	197	187	184	99	90
24.	-	188	-	190	-	-	-	78	-
25.	200	-	285	300	271	-	257	127	-
26.	135	218	214	241	226	214	180	90	-
27.	-	252	284	300	271	-	255	105	95
28.	210	-	262	264	-	-	-	100	95
29.	135	200	230	232	210	198	180	100	96
30.	-	-	235	222	197	-	-	-	-
31.	--	190	245	261	240	-	-	-	-
32.	174	200	214	223	193	184	182	90	87
33.	-	231	246	246	226	-	-	90	-

Cont....

34.	-	-	-	228	-	-	-	96	91
35.	145	235	252	265	260	246	230	-	-
36.	-	-	247	246	210	-	-	-	-
37.	-	-	230	236	206	-	-	81	-
38.	200	256	257	272	250	218	200	-	119
39.	-	-	253	252	231	-	-	91	86
40.	-	205	244	245	221	-	-	90	86
41.	201	288	285	285	271	242	231	-	-
42.	-	-	260	251	229	-	-	93	87
43.	-	-	-	209	282	-	-	90	-
44.	139	220	232	280	271	230	225	-	-
45.	-	-	248	248	-	-	171	86	-
46.	--	-	-	200	196	-	-	80	-
47.	162	200	212	220	193	184	180	-	-
48.	-	-	242	242	-	-	-	88	88
49.	-	-	240	251	220	-	-	91	-
50.	132	201	236	258	223	228	190	86	-
51.	-	222	240	240	219	212	-	86	81
52.	141	-	-	230	204	-	-	87	83
53.	170	230	258	262	238	214	205	114	-
54.	-	220	230	231	212	206	-	90	90
55.	-	216	237	237	220	211	-	103	-
56.	205	212	257	261	232	222	206	90	87
57.	-	198	210	240	213	-	-	80	78
58.	-	230	246	253	230	-	222	-	-
59.	130	190	193	261	240	220	210	115	108
60.	170	225	242	246	218	210	208	-	-

h = Hour d = Day m = month

CBP = Cord blood from placental end.

CBF = Cord blood from foetal end.

2. Serum Triglycerides (STG) (mg%dl).

Sl. No.	Trimester I	Trimester II	Trimester III	Intra-partum	Post partum 24h	Post partum 7d	Post partum 1m	CBP	CBF
1.	-	91	-	100	91	82	80	-	-
2.	-	-	105	-	90	94	92	-	-
3.	110	115	-	120	-	103	-	33	-
4.	95	98	118	-	-	-	-	-	-
5.	85	78	142	140	115	102	100	65	-
6.	100	-	105	-	100	-	-	-	59
7.	68	-	76	-	90	-	86	-	-
8.	90	98	95	112	107	101	87	41	-
9.	84	85	86	105	-	80	-	-	-
10.	110	125	140	130	-	-	130	-	-
11.	92	90	145	112	127	119	124	-	50
12.	-	-	-	108	105	-	-	-	-
13.	-	-	-	110	106	100	-	-	-
14.	94	90	130	140	115	110	104	40	-
15.	95	101	-	102	-	92	-	60	46
16.	85	-	-	90	-	85	70	-	-
17.	100	105	114	120	111	110	105	40	36
18.	-	88	-	100	-	91	-	37	-
19.	-	-	-	96	90	-	-	34	-
20.	120	116	140	120	115	100	108	50	-
21.	94	97	99	100	-	-	-	45	-
22-	-	-	-	100	90	90	-	-	-
23.	92	98	100	113	107	105	102	48	43
24.	-	90	-	98	-	-	-	33	-
25.	112	-	144	152	120	-	116	63	-
26.	98	100	98	130	118	110	82	44	-
27.	-	130	150	162	140	-	127	50	41
28.	102	-	126	140	-	-	-	47	45
29.	92	85	115	118	116	100	91	50	45
30.	-	-	112	116	94	-	-	-	-
31.	-	89	140	148	136	-	-	-	-
32.	92	95	108	112	114	106	100	41	36
33.	-	124	133	132	110	-	-	41	-

Cont....

34.	–	–	–	107	–	–	–	45	43
35.	101	125	136	132	128	114	122	–	–
36.	–	–	122	125	112	–	–	–	–
37.	–	–	120	122	110	–	–	35	–
38.	108	118	135	140	124	98	90	–	–
39.	–	–	122	121	110	–	–	41	34
40.	–	90	108	106	96	–	–	43	40
41.	92	94	110	128	121	145	108	–	–
42.	–	–	130	120	108	–	–	46	41
43.	–	–	–	94	86	–	–	40	–
44.	86	124	131	167	112	101	98	–	–
45.	–	–	121	120	–	–	–	46	46
46.	–	–	–	99	97	–	–	40	–
47.	96	92	112	114	104	90	90	–	–
48.	–	–	115	116	–	–	–	42	42
49.	–	–	123	126	104	–	–	35	–
50.	85	96	124	135	110	110	105	34	–
51.	–	96	117	118	110	102	–	43	40
52.	90	–	–	116	109	–	–	33	30
53.	100	100	128	140	109	98	97	60	–
54.	–	101	120	120	116	110	–	–	–
55.	–	96	115	116	114	101	–	35	35
56.	95	110	115	132	130	128	126	48	–
57.	–	91	101	112	104	–	–	48	45
58.	–	115	140	142	132	–	118	45	44
59.	86	89	90	148	136	125	119	–	–
60.	104	114	107	124	100	98	61	59	–

3. HDL Cholesterol (mg/dl)

Sl. No.	Trimester I	Trimester II	Trimester III	Intra-partum	Post partum 24h	Post partum 7d	Post partum 1m	CBP	CBF
1.	-	38	-	41	39	39	37	-	-
2.	-	-	42	-	36	35	34	-	-
3.	33	42	-	44	-	38	-	17	-
4.	30	37	44	-	-	-	-	-	-
5.	30	34	46	44	42	40	37	20	-
6.	30	-	38	-	41	-	-	-	-
7.	32	-	40	-	41	-	37	-	-
8.	30	40	41	52	46	43	34	19	-
9.	28	36	33	38	-	32	-	-	-
10.	32	40	42	45	-	-	41	-	-
11.	30	33	46	40	44	40	40	-	19
12.	-	-	-	39	44	-	-	-	-
13.	-	-	-	42	41	42	-	-	-
14.	28	35	44	46	41	41	40	20	-
15.	30	38	-	42	-	39	-	20	20
16.	28	-	-	34	-	32	30	-	-
17.	30	40	45	51	46	45	36	20	19
18.	-	33	-	41	-	40	-	16	-
19.	-	-	-	39	39	-	-	22	-
20.	35	40	45	41	38	42	42	18	-
21.	29	36	30	39	-	-	-	-	-
22.	-	-	-	40	38	38	-	-	-
23.	30	32	35	56	54	48	48	18	18
24.	-	33	-	36	-	-	-	16	-
25.	33	-	45	45	41	-	41	22	-
26.	33	36	40	49	49	39	31	20	-
27.	-	41	45	46	42	-	40	18	16
28.	32	-	44	45	-	-	-	19	19
29.	30	34	48	48	47	34	32	20	19
30.	-	-	44	40	36	-	-	-	-
31.	-	35	45	45	42	-	-	-	-
32.	35	36	50	44	44	40	39	17	16
33.	-	39	41	42	43	-	-	18	-

Contd.....

34.	-	-	-	42	-	-	-	19	19
35.	30	41	49	40	41	42	40	-	-
36.	-	-	48	42	39	-	-	-	-
37.	-	-	46	46	43	-	-	18	-
38.	37	42	45	45	40	42	32	-	21
39.	-	-	53	49	43	-	-	20	20
40.	-	35	42	42	37	-	-	19	19
41.	36	38	45	45	45	44	38	-	-
42.	-	-	50	48	43	-	-	20	19
43.	-	-	-	39	38	-	-	19	-
44.	30	42	46	47	44	42	36	-	-
46.	-	-	-	42	42	-	-	20	20
47.	32	35	46	50	50	40	38	-	-
48.	-	-	44	45	-	-	-	20	20
49.	-	-	42	43	40	-	-	19	-
50.	30	42	44	46	42	40	38	17	-
51.	-	36	50	54	50	39	-	21	20
52.	33	-	-	49	45	-	-	20	20
53.	28	39	44	45	37	35	32	21	-
54.	-	35	47	47	40	39	-	-	-
55.	-	39	47	48	44	38	-	19	19
56.	32	43	41	50	46	40	38	19	-
57.	-	42	48	54	43	-	-	20	18
58.	-	43	50	51	40	-	38	21	19
59.	28	35	35	45	42	39	37	-	-
60.	28	38	41	42	40	40	38	19	18

ABBREVIATIONS USED

STC : Serum Total Cholesterol

STG : Serum Triglycerides

HDL : High density lipoprotein cholesterol

LDL : Low density lipoprotein cholesterol

VLDL : Very low density lipoprotein ~~cholesterol~~

PP : Post partum

IP : Intrapartum

d : Days

h : Hour

m : Month

PP24h: 24hours postpartum

PP7d : 7 days post partum

PP1m : 1 month postpartum

CBP : Cord blood placental end.

CBF : Cord blood foetal end.

S.eco.: Socio_economic status.

V.D.: Vaginal delivery

Em.CS : Emergency caesarean section.

Elec.CS: Elective caesarean section

Lact. : Lactating

N.Lact.: Non-lactating

L : Low

M : Middle

Veg. : Vegetarian

N.V. : Non-vegetarian
